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Fatherhood at young age linked to greater likelihood of mid-life death

Becoming a dad before age of 25 seems to pose greatest risk, suggests research

Becoming a dad before the age of 25 is linked to a heightened risk of dying early in middle age, indicates a sibling study published online in the Journal of Epidemiology & Community Health.

The published evidence suggests that men who father a child in early life have poorer health and die earlier than men who delay fatherhood, but family environment, early socioeconomic circumstances and genes are thought to explain this association.

In a bid to tease out the underlying factors, the researchers used a 10 per cent nationally representative sample of households drawn from the 1950 Finnish Census. This involved more than 30,500 men born between 1940 and 1950, who became fathers by the age of 45. The dads were tracked from the age of 45 until death or age 54, using mortality data for 1985-2005.

Some 15% of this sample had fathered their first child by the age of 22; 29% at ages 22-24; 18% when they were 25-26; 19% between the ages of 27 and 29; and 19% between the ages of 30 and 44. The average age at which a man became a dad was 25-26, and men in this age bracket were used as a reference.

The researchers took account of influential factors, such as educational attainment and region of residence, which are linked to the timing of first parenthood; and marital status and number of children, both of which are linked to long term health.

During the 10 year monitoring period around 1 in 20 of the dads died. The primary causes of death were ischaemic heart disease (21%) and diseases related to excess alcohol (16%).

Men who were dads by the time they were 22 had a 26% higher risk of death in mid-life than those who had fathered their first child when they were 25 or 26. Similarly, men who had their first child between the ages of 22 and 24 had a 14% higher risk of dying in middle age.

These findings were independent of factors in adulthood or year of birth.

At the other end of the scale, those who became dads between the ages of 30 and 44 had a 25% lower risk of death in middle age than those who fathered their first child at 25 or 26. The risk of death for men fathering their first child between the ages of 27 and 29 was the same as that of men in the reference group.

In a subsidiary sample of 1124 siblings, brothers who had become dads by the age of 22 were 73% more likely to die early than their siblings who had fathered their

first child at the age of 25 or 26. Similarly, those who entered parenthood at 22-24 were 63% more likely to die in mid life. These findings were held true, irrespective of year of birth, shared early life circumstances, educational attainment, marital status, region of residence, and number of children.

Once again, men who became dads between the ages of 30 and 44 had a 22% lower risk of a mid-life death, although this was statistically the same as those who fathered their first child at 25/26.

"The findings of our study suggest that the association between young fatherhood and mid life mortality is likely to be causal," write the researchers.

"The association was not explained by unobserved early life characteristics shared by brothers or by certain adult characteristics known to be associated both with fertility timing and mortality," they explain.

They go on to say that although having a child as a young adult is thought to be less disruptive for a man than it is for a woman, taking on the combined role of father, partner and breadwinner may cause considerable psychological and economic stress for a young man and deprive him of the ability to invest in his own wellbeing.

he researchers point out that while these factors may not be so important for today's generation of dads, they may nevertheless experience other types of stressors.

"The findings of our study provide evidence of a need to support young fathers struggling with the demands of family life in order to promote good health behaviours and future health. The promotion of good health behaviours in young fathers could also support healthy behaviour in their children," they suggest.

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Scientists identify that memories can be lost and found

Memories are more robust than we thought

A team of scientists believe they have shown that memories are more robust than we thought and have identified the process in the brain, which could help rescue lost memories or bury bad memories, and pave the way for new drugs and treatment for people with memory problems.

Published in the journal Nature Communications a team of scientists from Cardiff University found that reminders could reverse the amnesia caused by methods previously thought to produce total memory loss in rats. .

"Previous research in this area found that when you recall a memory it is sensitive to interference to other information and in some cases is completely wiped out.

"Our research challenges this view and we believe proves this not the case," according to Dr Kerrie Thomas, who led the research.

"Our research found that despite using a technique in the brain thought to produce total amnesia we've been able to show that with strong reminders, these memories can be recovered,"

Whilst the results were found in rats, the team hope it can be translated into humans and new drugs and treatments could be developed for people suffering with memory disorders. Dr Thomas added: "We are still a very long way off from helping people with memory problems.

"However, these animal models do accurately reflect what's happening in humans and suggest that our autobiographical memories, our self-histories, are clouded by new memories rather than actually lost. This is an exciting prospect in terms of treating psychiatric illness associated with memory disorders such as post-traumatic stress disorder, schizophrenia and psychosis.

"We can now devise new drugs or behavioural strategies that can treat these memory problems in the knowledge that we won't overwrite our experiences," she added.

http://www.eurekalert.org/pub_releases/2015-08/rumc-aaa080415.php

Eating away at cognitive decline

MIND diet may slow brain from aging by 7.5 years

While cognitive abilities naturally diminish as part of the normal aging process, it may be possible to take a bite out of this expected decline.

Eating a group of specific foods known as the MIND diet may slow cognitive decline among aging adults, even when the person is not at risk of developing Alzheimer's disease, according to researchers at Rush University Medical Center. This finding is in addition to a previous study by the research team that found that the MIND diet may reduce a person's risk in developing Alzheimer's disease.

The recent study shows that older adults who followed the MIND diet more rigorously showed an equivalent of being 7.5 years younger cognitively than those who followed the diet least.

The results of the study recently were published online in the journal *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*.

Mediterranean, with a DASH of other ingredients

The National Institute of Aging funded study evaluated cognitive change over a period of 4.7 years among 960 older adults who were free of dementia on enrollment. Averaging 81.4 years in age, the study participants also were part of the Rush Memory and Aging Project, a study of residents of more than 40 retirement communities and senior public housing units in the Chicago area.

During the course of the study, they received annual, standardized testing for cognitive ability in five areas - episodic memory, working memory, semantic

memory, visuospatial ability, and perceptual speed. The study group also completed annual food frequency questionnaires, allowing the researchers to compare participants' reported adherence to the MIND diet with changes in their cognitive abilities as measured by the tests.

Martha Clare Morris, ScD, a nutritional epidemiologist, and colleagues developed the diet, whose full name is the Mediterranean-DASH Diet Intervention for Neurodegenerative Delay. As the name suggests, the MIND diet is a hybrid of the Mediterranean and DASH (Dietary Approaches to Stop Hypertension) diets. Both diets have been found to reduce the risk of cardiovascular conditions, like hypertension, heart attack and stroke.

"Everyone experiences decline with aging; and Alzheimer's disease is now the sixth leading cause of death in the U.S., which accounts for 60 to 80 percent of dementia cases. Therefore, prevention of cognitive decline, the defining feature of dementia, is now more important than ever," Morris says. "Delaying dementia's onset by just five years can reduce the cost and prevalence by nearly half."

A wine and no cheese party

The MIND diet has 15 dietary components, including 10 "brain-healthy food groups" and five unhealthy groups - red meat, butter and stick margarine, cheese, pastries and sweets, and fried or fast food.

To adhere to and benefit from the MIND diet, a person would need to eat at least three servings of whole grains, a green leafy vegetable and one other vegetable every day -- along with a glass of wine -- snack most days on nuts, have beans every other day or so, eat poultry and berries at least twice a week and fish at least once a week.

In addition, the study found that to have a real shot at avoiding the devastating effects of cognitive decline, he or she must limit intake of the designated unhealthy foods, especially butter (less than 1 tablespoon a day), sweets and pastries, whole fat cheese, and fried or fast food (less than a serving a week for any of the three).

Berries are the only fruit specifically to be included in the MIND diet. "Blueberries are one of the more potent foods in terms of protecting the brain," Morris says, and strawberries also have performed well in past studies of the effect of food on cognitive function.

"The MIND diet modifies the Mediterranean and DASH diets to highlight the foods and nutrients shown through the scientific literature to be associated with dementia prevention," Morris explains.

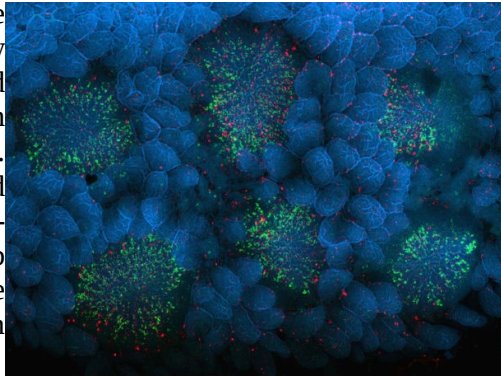
"There is still a great deal of study we need to do in this area, and I expect that we'll make further modifications as the science on diet and the brain advances."

http://www.eurekalert.org/pub_releases/2015-08/uoe-bis080415.php

Brain infection study reveals how disease spreads from gut

Research could enable earlier detection of prion diseases

Diagnosis of deadly brain conditions could be helped by new research that shows how infectious proteins that cause the disease spread. The study reveals how the proteins - called prions - spread from the gut to the brain after a person or animal has eaten contaminated meat. Scientists say their findings could aid the earlier diagnosis of prion diseases - which include variant Creutzfeldt-Jakob disease (vCJD) in people and bovine spongiform encephalopathy (BSE) in cows.



This magnified image of the small intestine shows specialized structures called Peyer's patches -- which are part of the body's immune system. Researchers have shown how deadly proteins called prions -- which cause variant CJD in people and BSE in cows -- invade the body through Peyer's patches before infecting the brain. Prof Neil Mabbott & Dr David Donaldson, The Roslin Institute, University of Edinburgh

In people, the disease remains very rare - 229 people have died from vCJD since it was first identified almost 20 years ago, of which 177 were from the UK.

Prions are infectious proteins with abnormal shapes that can be passed between people and animals by eating contaminated meat. Until now, it was not known how prions spread from the gut to the brain after consuming infected meat.

Researchers at University of Edinburgh's Roslin Institute studied the course of prion infection in mice.

They found that prions must first build up in specialised structures in the lining of the small intestine before they are able to spread throughout the body to the brain. The structures - called Peyer's patches - are part of the body's immune system and form the first line of defence against contaminated food. The study suggests prions hijack Peyer's patches to cause infection.

Prions did not build up in similar patches in the large intestine until a later stage of infection, the team found. At this stage, prions were also detected in the spleen and lymph nodes.

As many as one in 2000 people in the UK could be carrying infectious prions without showing any symptoms of disease, according to recent estimates. These are based on analysis of tissue taken during routine appendix removal operations.

The researchers say that these estimates may fail to identify individuals in the earliest stages of infection, where prions have not yet spread beyond the small intestine.

When prions get into the brain, they destroy nerve cells. This can lead to major neurological symptoms including memory impairment, personality changes, and difficulties with movement.

Other prion diseases include scrapie in sheep and chronic wasting disease in deer. Professor Neil Mabbott, of The Roslin Institute at the University of Edinburgh, who led the study, said: "Whether all individuals with evidence of prion infection in their gut go on to develop neurological disease is not known. We need a greater understanding of what factors enhance our susceptibility to prion diseases so that we can put in place safeguards to prevent these conditions from spreading in people and farmed animals."

The study, published in Journal of Virology, was funded by the Biotechnology and Biological Sciences Research Council.

<http://bit.ly/1L1dlF>

Swedish Designers Are Turning Fruits and Veggies Into a Nonperishable Powder

The dried and powdered produce, called FoPo, could become a staple in disaster relief

When you toss manky lettuce or moldy berries think about this: Globally, according to the Food and Agriculture Organization, we waste [more than a third](#) of the food we produce.

To combat that, a group of Swedish graduate students in the Food Innovation and Product Design program at Lund University have come up with a way to use produce that is about to go to waste—and to help people who have limited access to food.

They're calling it [FoPo Food Powder](#), and it's exactly what it sounds like: dried, powdered, shelf-stable fruits and vegetables, which can be dropped into relief efforts after natural disasters or distributed in low-resource areas where fresh food and refrigeration are both hard to come by. "When we found out that one third of the food produced was going to waste while people in the world were starving we couldn't back out," says Kent Ngo, one of the students who developed it.

Ngo says they're not producing something revolutionary—powdered food has been around since the early days of astronauts—but that they're rethinking the waste and distribution channels. While their development team reached out to farmers and retailers to source fruit, the food scientists experimented with different drying and powdering techniques. They settled on spray-drying it, then

grinding it up after it was sublimated. From there, they looked at ways to distribute it, through commercial and government supported venues.

The makers of FoPo have experimented with several different kinds of fruits. (FoPo Food Powder)

One member of the group, Gerald Perry Marin, grew up in the Philippines, so he'd seen how typhoons and other natural disasters cut people off from their food supply, and how important it was to have food options that were easy to access in a relief scenario.

"Today a relief bag for humanitarian disasters contains various foods such as strawberry jam, peanut butter and peas in tomato sauce. We think that an easily transported pack of cheap dried food powder with high nutritional value would fit in perfectly," Ngo says. The team has been trying to keep its prices down, too, to aid low-budget humanitarian groups and NGOs.

Freeze-dried food retains most of the nutritional benefits of raw food. It loses some vitamin and mineral density in the drying process, but it's still a good way to get fiber and nutrients.

The makers of FoPo are currently running a pilot program in Manila. For their first run, they're drying calamansi, a citrus fruit that Ngo says tastes like a mix of lime and tangerine. There is a surplus of it, it's not available in other places, and it is easy for their Philippine manufacturing program to dry and powder.

The group has reportedly gotten support from senators in the Philippines, and they're about to start working with the UN's [Initiative on Food Loss and Waste](#), to try to reach more people and countries that could benefit. To broaden their reach, they're also working with commercial distributors and manufacturers that want to use FoPo in their food products, like cake mixes and ice cream. Consumers can also sprinkle it into food or drinks, or use it in baking. The company has almost 40 international supermarkets on board.

"I was a bit surprised that the calamansi powder tasted so good," Ngo says. "I can't wait for the mango and pineapple powder."

<http://www.bbc.com/news/science-environment-33772541>

Balancing rocks trace history of 'jumping' earthquakes

The researchers spent 10 years collecting measurements of balancing rocks

By Jonathan Webb Science reporter, BBC News

US scientists say they have solved the riddle of why a collection of balancing rocks near the San Andreas fault has never been toppled by earthquakes.

Their decade-long study concludes that quakes can stop or "jump" due to interactions between the San Andreas and the neighbouring San Jacinto fault.

Models show that these interactions sent the biggest vibrations around the rock stacks, leaving them intact.

But the connected nature of the faults has implications for quake planning. The study of precariously balanced rocks was begun in the 1990s by Jim Brune, now an emeritus professor at the University of Nevada and a co-author of the new paper. "He realised that [these rocks] could be a check on seismic hazard maps, and give long-term indications of ground shaking," said the study's lead author Prof Lisa Grant Ludwig, from the University of California, Irvine.

"They are kind of natural seismoscopes - but you have to read them indirectly.

"They don't tell you an earthquake happened, they tell you 'an earthquake strong enough to knock me down did not happen'."

Precarious rocks, like this one in Nevada, can act as natural measures of earthquake strength over time

Tippling point

Generally, balancing rocks are not seen within 15km of major faults. But 10 years ago Prof Brune and his colleagues found two sizeable collections of such stones just 7-10km from the San Andreas and San Jacinto faults, in the San Bernardino mountains of California.

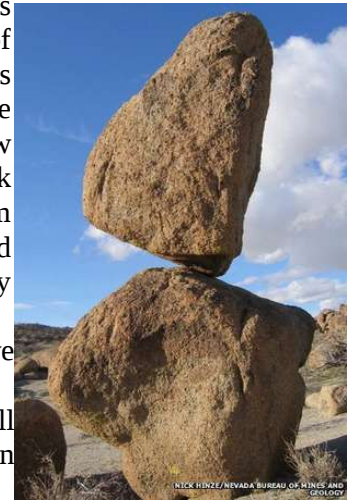
In the new study, due to be published in the journal *Seismological Research Letters*, these rocks were carefully catalogued and measured.

Importantly, the team calculated how much force it would take to tip each of the rocks over. "There are two methods of doing that, one of which is actually trying to tip the thing," Prof Ludwig said. This meant some nerve-racking fieldwork, gently pushing the rocks until there was some movement, but not actually tipping them over.

"If my mother had known I was doing that, she would not have been happy," Prof Ludwig confessed. "You never want to be on the downhill side when you tip it."

The second method, for rocks too dangerous or difficult to tip, was "photomodelling": using views from multiple angles to build a 3D model of the balanced stone and calculate its centre of gravity, mass, and so on. Both these methods, along with some "shake table" simulation experiments, showed that the rocks should have fallen over during quakes as recent as 1812 and 1857.

But various measures can tell us exactly how long the stones have perched in their places - and it is millennia, not centuries. "One of my former postdocs did an age



study of one of the rocks. And it'd been in that position about 18,000 years," said Prof Ludwig.

So how did these precarious rocks withstand the tens or hundreds of earthquakes that shook the region during that time?

Network of fractures

"The inescapable conclusion was that the ground motions had to be lower than you would expect from typical earthquakes on the San Andreas and San Jacinto faults," Prof Ludwig explained. The team's best explanation for that surprisingly small ground movement - and one supported by computer modelling of big earthquakes - is an interaction between the two faults.

"The San Andreas and San Jacinto faults come very close together; they're only about 2km apart. And it's been well established, through other earthquakes and modelling studies, that a rupture can jump across [a gap like that]. It's what's called a stepover.

"What if the rupture jumped across, or alternatively, stopped at this junction, or started at this junction? All three of those cases would produce lower ground shaking in the area where we found the rocks."

It is crucial to consider the faults together, Prof Ludwig said - not just to explain the baffling, balancing rocks, but also in order to plan safely for future earthquakes. "These are really networks of fractures in the earth. Just because we give them different names doesn't mean that they behave independently."

Shaky scenario

Dr Lucy Jones is a long-serving seismologist and a science adviser for risk reduction at the US Geological Survey. She said the paper would have "pretty significant implications" for earthquake planning in California.

In particular, Dr Jones said the findings might impact the "ShakeOut scenario" - in which she and others modelled a major San Andreas quake, to support safety drills and procedures.

"I think that this study actually makes the particular ShakeOut scenario less likely, but I'm not sure it means that we're definitely going to get less ground motion," Dr Jones told the BBC.

"It isn't a clear-cut answer as to whether we'll be better off or worse off. We're going to need time to look at the permutations."

Looking beyond individual quakes, Dr Jones said the new study fits into a "pretty well accepted picture" that in the long-term, seismic activity is gradually shifting from the southern stretch of the San Andreas fault across to the younger San Jacinto fault.

"This study is a really cool piece of evidence that maybe the jump is a little further along than we assumed," she said.

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Penn scientists identify key genetic factor that keeps moles from turning into melanoma

Research also yields realistic new model of melanoma for testing therapies

PHILADELPHIA -- Moles are benign tumors found on the skin of almost every adult. Scientists have known for years that a mutation in the BRAF gene makes them start growing, but until now haven't understood why they stop. Now, researchers from the Perelman School of Medicine at the University of Pennsylvania have identified a major genetic factor that keeps moles in their usual non-cancerous, no-growth state. The study was published online first this summer in the journal *Cancer Discovery*.

"The BRAF mutation that stimulates the initial growth of moles also stimulates the production of a tumor suppressor protein, p15, which ultimately acts as a powerful brake on further cell division," said senior author Todd W. Ridky MD, PhD, an assistant professor of Dermatology at Penn. "It's this cell division that ultimately allows the transition from a normal mole into melanoma. When mole cells lose the p15 brake, cells can start dividing again and can progress into cancer."

For their study, Ridky and his colleagues developed a new model of human melanoma, using tissue engineering to make skin grafts containing human mole cells in which p15 was removed. When combined with other mutations known to be important for the development of melanoma, and transplanted into mice, the p15 depleted cells progressed into melanoma.

"The model tissues are medically relevant because they used the naturally occurring human mole cells in the 3-dimensional environment of living skin, which allows detailed functional studies - the field hasn't had an experimental system like this before," said lead author Andrew McNeal, a research specialist in Ridky's lab.

How Moles are Born

Both moles and melanomas originate from melanin-producing cells (melanocytes) within the skin. Scientists have known for more than a decade that one particular mutation is responsible for the abnormal melanocyte growth that creates the majority of both benign moles and cancerous melanomas. The mutation, in a cell-growth gene called BRAF, causes BRAF to be in an "always on" state, continuously promoting cell division.

In moles, however, cell proliferation typically stops after the cluster of melanocytes has reached the few millimeters (or roughly the size of a pencil eraser). "Why moles stop growing, despite all that BRAF activity, has been a

long-standing question in the field," Ridky said. "To answer that question, Ridky and colleagues studied mole cells isolated directly from normal benign moles removed from patients, and compared them to melanocytes isolated from normal (non-mole) skin. The mole melanocytes had 140 times more p15 than the normal skin melanocytes.

Comparing cells from patient melanomas that had originated from previously benign moles, the researchers found generally high p15 levels in the mole tissue, and very low or undetectable p15 in the melanomas. This suggested that p15 is important for holding regular moles in a benign state, and that any subsequent loss of p15 would promote the transition to melanoma. Ridky and his team showed that the BRAF over-activation that drives the mole growth also causes the mole cells to secrete a signaling molecule called TGF- β , which in turn, signals back to the mole cells to make p15. These findings hinted at a possible explanation for the curious fact that most moles have to reach a diameter of at least a few millimeters before they stop growing - TGF β has to build up to a sufficient level first, and small collections of mole cells don't lead to enough local TGF β production in the mole to stop cell division.

An Overlooked Factor

The importance of p15 has been largely underappreciated up to now, said Ridky, because many researchers have assumed that a different, but related, tumor suppressor protein, p16, does the main work of growth-inhibition in moles. The gene for p16 is physically close to p15 in the nuclear DNA, is present in moles, and is also lost in melanomas and many other cancers. While the two tumor suppressors normally work together to keep the brakes on cell proliferation in moles, Ridky and his team found evidence suggesting that p15 has unique functions. For example, inserting p15 into normal cells was enough to halt proliferation completely, whereas inserting p16 only slowed proliferation.

"Clearly p15 is doing things that p16 doesn't, and that's something that the field has mostly overlooked," Ridky said.

Modeling Melanoma, and Beyond

Ridky now plans to experiment extensively with the model to provide insights into how melanoma develops, and how it might be targeted with new therapies.

He and his colleagues also will study p15's possible roles in other cancers. "That deletion that simultaneously takes out both p16 and p15 is among the most common DNA deletions in human cancers," Ridky said. "Cancer biologists have generally assumed that p16 is the more important of the two, but I think that we're going to find important and unique roles for p15 even beyond the context of moles and melanoma."

Other Penn authors on the study include Kevin Liu, Vihang Nakhate, Christopher A. Natale, Elizabeth K. Duperret, Brian C. Capell, Tzvete Dentchev, Shelley L. Berger, Meenhard Herlyn, and John T. Seykora.

Funding was provided by the National Institutes of Health (R01CA163566, R01CA165836, F31CA1186446, CA076674, CA182890, P01AG031862), the Melanoma Research Alliance, the Dermatology Foundation, the American Skin Association, and the Melanoma Research Foundation.

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Endoscopes still contaminated after cleaning, study shows

Bacteria can survive on endoscopes despite multi-step cleaning and disinfecting

Washington, DC - Potentially harmful bacteria can survive on endoscopes used to examine the interior of the digestive tract, despite a multi-step cleaning and disinfecting process, according to a study published in the August issue of the American Journal of Infection Control, the official publication of the Association for Professionals in Infection Control and Epidemiology (APIC).

Though endoscopes were cleaned in accordance with multi-society guidelines, viable microbes and residual contamination remained on surfaces after each stage of cleaning, according to study findings.

Researchers from Ofstead & Associates in Saint Paul, Minnesota and Mayo Clinic in Rochester, Minnesota tested samples collected from 60 encounters with 15 colonoscopes and gastroscopes used for gastrointestinal procedures after each reprocessing step to assess contamination levels. Investigators observed all reprocessing activities, using a checklist to ensure that cleaning protocols were performed in accordance with published guidelines.

Reprocessing consisted of: bedside cleaning, manual cleaning in dedicated reprocessing rooms, and automated endoscope reprocessing with a high-level disinfectant. Disinfected endoscopes were stored vertically after drying with isopropyl alcohol and forced air. When contamination levels exceeded pre-determined benchmarks for each cleaning step, technicians went beyond guidelines and repeated cleaning procedures, retesting after each attempt to reduce contamination.

Researchers performed microbial cultures and various rapid tests to detect viable organisms and organic residue that remained after each step of cleaning. Viable organisms were detected on 92 percent of devices after bedside cleaning; 46 percent after manual cleaning; 64 percent after high-level disinfection, and 9 percent after overnight storage. Rapid indicator tests detected contamination above benchmarks on 100 percent of devices after bedside cleaning; 92 percent after manual cleaning; 73 percent after high-level disinfection, and 82 percent after overnight storage.

"This study demonstrates that colonoscopes and gastroscopes can harbor residual organic material, including viable microbes, even when adherence with recommended reprocessing guidelines is verified," said the study authors. "More research is needed to identify processes that can ensure all flexible endoscopes are free of residual contamination and viable microbes prior to patient use, including the potential use of routine monitoring with rapid indicators and microbiologic cultures. Results from this study suggest that current standards and practices may not be sufficient for detecting and removing residual contamination."

The authors list several potential limitations of the study including that it is a single-site study and may not be generalizable nationwide. In addition, reprocessing technicians were aware of the researchers' use of a checklist to ensure guideline compliance and therefore may have devoted more time and effort to reprocessing. Another caveat is that technicians were immediately informed about contamination that exceeded benchmarks and repeated cleaning steps--actions that are not generally part of standard practice.

Colonoscopes and gastroscopes are endoscopic devices with thin tubes, channels, and ports that are used to examine the interior of the colon and the stomach. Recent reports of multidrug-resistant infections related to contaminated duodenoscopes, which have intricate elevator mechanisms and channels that are especially difficult to clean, have raised awareness about the necessity for meticulous reprocessing of all types of endoscopes to prevent the transmission of pathogens to patients.

http://www.eurekalert.org/pub_releases/2015-08/iu-rfr080515.php

Researchers find romantic kissing is not the norm in most cultures

Romantic kissing not the norm in most cultures, some find it repulsive

BLOOMINGTON, Ind. -- For generations, passionate kisses immortalized in movies, songs and the arts have served as a thermometer of romantic affection.

But current research has found that not only is romantic kissing not the norm in most cultures, some find it uncomfortable and even flat-out repulsive.

Justin Garcia, research scientist at Kinsey Institute at Indiana University, is the co-author of a new study published in the journal *American Anthropologist* -- "Is the Romantic-Sexual Kiss a Near Human Universal?" -- that looked at 168 cultures throughout the world to better understand where kissing does and doesn't occur.

Using standard cross-cultural methods, the study found that fewer than half of all cultures surveyed -- 46 percent -- engage in romantic/sexual kissing. Romantic kissing was defined as lip-to-lip contact that may or may not be prolonged.

"We hypothesized that some cultures would either not engage in romantic/sexual kissing, or find it to be a strange display of intimacy, but we were surprised to find that it was a majority of cultures that fell into this category," said Garcia, assistant professor of gender studies in the IU Bloomington College of Arts and Sciences. "This is a real reminder of how Western ethnocentrism can bias the way we think about human behavior."

Romantic kissing was most prevalent in the Middle East, where all 10 of the cultures studied engaged in it. In North America, 55 percent of cultures engaged in romantic kissing, along with 70 percent in Europe and 73 percent in Asia. But there was no evidence of romantic kissing in Central America, and no ethnographer working with Sub-Saharan African, New Guinean or Amazonian foragers or horticulturalists reported any evidence of romantic kissing in the populations they studied, according to the research.

The research conducted by Garcia and colleagues also found a relationship between social complexity and kissing: The more socially complex and stratified a society is, the higher the frequency of romantic kissing.

Interest in the study stemmed from renewed attention in the role of close touch and kissing in people's romantic and sexual lives, Garcia said. Recent work on the issue, he said, has made claims about the universality of erotic kissing, some even claiming 90 percent of societies engage in the act.

"However, we realized no one had used standard cross-cultural methods to assess how frequently kissing actually occurs in different societies, but by doing so, we could begin to understand why it might occur in some places and not others," he said.

It is not clear where romantic/sexual kissing evolved from, Garcia said. Some animals engage in similar behaviors; chimpanzees, for example, are known to engage in open-mouth kissing. When it comes to humans kissing, Garcia pointed out that it does serve as a way to learn more about a partner, "whether one feels there is any 'chemistry,' or possibly to assess health via taste and smell, and in some ways to assess compatibility with each other."

"There is likely a biological underpinning to kissing, as it can often involve exchange of pheromones and saliva, and also pathogens -- which might be particularly dangerous in societies without oral hygiene, where kissing may lead to spread of respiratory or other illness," he said. "But this is only in societies that have come to see the erotic kiss as part of their larger romantic and sexual repertoires. How that shift occurs is still an open question for research."

Study co-authors are William Jankowiak, Distinguished Professor of Anthropology, and Shelly Volsche, graduate research assistant in anthropology, both at the University of Nevada, Las Vegas.

<http://bit.ly/1J35hOQ>

Why This Chicken is Black From Comb to Feathers to Muscles

The Ayam Cemani is the Goth of the chicken world

By [Marissa Fessenden](#) smithsonian.com

It's one of the rarest breeds of chicken in the United States, and also one of the priciest — [Greenfire Farms](#) sells a single day-old chick for a whopping \$199, and in 2013 aficionados of the unusual chicken could purchase [a juvenile pair for \\$4,999](#). Why the high price tag? It's all a matter of color: these fowl are black from the inside out.

[Kat McGowan reports](#) for *Nautilus* on the breed, called [Ayam Cemani](#). They have deep bluish-black combs, beaks, cheeks, skin and even a black tongue. The pigmentation continues on to the muscles and internal organs. "It is one Goth chicken," she writes.

A ban on imported live chickens from Indonesia keeps the Ayam Cemani rare in the U.S. But a few other chickens have this black pigmentation in their muscles and organs (a phenomenon called fibromelanosis), including Swedish [Bohuslän-Dals svarthöna](#), Vietnamese Black H'Mong, and the [Silkie](#), originally from China and flaunting feathers so fine they look like silky hair.

How exactly do those birds get their inky color? McGowan reports on [work](#) by Swedish, American and Chinese researchers to figure out why.

The fibromelanosis in all four breeds starts with changes in the developing embryo.

Pigment-producing cells called melanocytes normally travel through embryos and end up only in the skin and eyes.

But in these all-black birds, a mutation tells melanocytes to move to tissues that will become fibers as the body develops. Dark pigment ends up in the tissue connecting muscles together, as well as the fiber that holds organs together.

McGowan writes:

This genetic fluke is the result of two sizable chunks of DNA that are duplicated within the chromosome (one of them upside-down). Inside those stretches, Andersson's group also pinpointed a gene called endothelin-3 (EDN3), known to be involved in the regulation of pigment-producing melanocyte cells. About 10 times as much EDN3 was expressed in the skin of adult black chickens than in other breeds.

The wide geographic spread of these black birds is a testament to exactly how fascinating humans find the result of this mutation.

Though they hail from China, Vietnam, Sweden and Indonesia, all the chickens have the same mutation, writes McGowan. But do they all share a love of The Cure?

<http://bit.ly/1IF9ir9>

Spicy Food Linked to Lower Risk of Death

In a study of nearly half a million volunteers in China, those who ate chilies just a couple times a week had a 10 percent lower risk of death. Christopher

Intagliata reports

Chinese cuisine has a lot of [blazingly spicy dishes](#), like mapo tofu and hot pot. And, of course, there's the ever-present chili oil. "I like spicy food myself." Lu Qi, an epidemiologist at the Harvard School of Public Health. "Almost every day, I eat spicy food." That spicy tradition served as scientific inspiration for Qi: he and colleagues performed a study which found that [a daily dose of chilies](#) might actually be a boon to your health.

The researchers enrolled nearly half a million Chinese volunteers, aged 30 to 79. They quizzed them on their affinity for fiery foods, and followed each study subject for an average of seven years. During that time, more than 20,000 of the subjects died. But after controlling for factors like smoking history and income, the scientists found that the risk of death was 10 percent lower in those who ate spicy food a couple times a week, compared with those who abstained. And *daily* chili eaters, like Lu, had a 14 percent lower risk of dying. That figure held true for both men and women. And yes, while it is a relatively modest effect, Lu says to keep in mind: we're just talking about chilies here. "It's not medicine." The findings appear in the *British Medical Journal*. [Jun Lv et al, [Consumption of spicy foods and total and cause specific mortality: population based cohort study](#)]

Previous studies have shown that capsaicin, the active ingredient in chillies, has anti-inflammatory and antimicrobial effects, among others. But before you grab the Tabasco, this study does not prove causation. For example, could be that people with weaker constitutions avoid spicy foods—making chili lovers appear more hardy in comparison. And the authors do not recommend starting a chili habit if you already have a sensitive stomach. But at least one thing's clear: indulging in spicy food probably won't hurt. Other than, well, your tongue.

http://www.eurekalert.org/pub_releases/2015-08/uoew-080615.php

Why the human heart cannot regenerate

FAU researchers discover endogenous process that controls reproduction of cardiac muscle cells and may lead to new treatments for heart attacks and cancer

The results of their research have recently been published in the high-profile journal *eLife**

The ability of most cardiac muscle cells to reproduce disappears in humans and all other mammals shortly after birth. What remains unclear, however, is how this

happens and whether it is possible to restore this ability and therefore to regenerate the heart.

FAU researchers Dr. David Zebrowski and Prof. Dr. Felix B. Engel from the Department of Nephropathology at Universitätsklinikum Erlangen's Institute of Pathology and their colleagues have now found a possible explanation for this phenomenon. 'In our study we discovered that the centrosome in cardiac muscle cells undergoes a process of disassembly which is completed shortly after birth,' Prof. Engel explains. 'This disassembly process proceeds by some proteins leaving the centrosome and relocating to the membrane of the cell nucleus in which the DNA is stored. This process causes the centrosome to break down into the two centrioles of which it is composed, and this causes the cell to lose its ability to reproduce.'

The centrosome is an organelle found in almost every cell. In recent years, experiments have shown that if the centrosome is not intact, the cell can no longer reproduce. This raised the key question to what extent centrosome integrity could be manipulated - such as in cancer where cells reproduce at an uncontrolled rate.

The FAU researchers have now investigated whether the state of centrosome integrity is regulated naturally in the animal kingdom in order to control the reproduction of certain cells.

A dramatic difference

'We were incredibly surprised to discover that the centrosome in the cardiac muscle cells of zebrafish and amphibians remains intact into adulthood,' says Dr. David Zebrowski, who has been studying centrosomes for five years. 'For the first time, we have discovered a significant difference between the cardiac muscle cells of mammals and those of zebrafish and amphibians that presents a possible explanation as to why the human heart cannot regenerate.'

The discovery that there is a natural process that regulates centrosome integrity in the cardiac muscle cells of mammals opens up a range of possibilities for future research. Firstly, this observation provides a new starting point for attempts to stimulate the reproduction of cardiac muscle cells in humans to regenerate the heart. At the same time, centrosome integrity can be examined in order to find adult cardiac muscle cells that may have retained their ability to reproduce, which may enable new forms of medical treatment. Finally, a detailed understanding of the mechanism could also help researchers to develop methods of inhibiting the uncontrolled growth of cancer cells.

The study on heart regeneration was carried out as part of the project CYDER (Cell Cycle in Disease and Regeneration) that was set up by Prof. Dr. Felix B. Engel, an expert in the field of heart regeneration. CYDER has been funded by FAU's Emerging Fields Initiative since 2014. EFI aims to promote outstanding,

preferably interdisciplinary research projects at an early stage and in a flexible and non-bureaucratic way. A strict selection process guarantees the high quality of the projects, approaches and the researchers funded within the scheme.

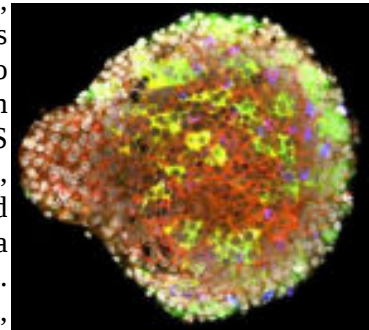
*The paper is available at <http://dx.doi.org/10.7554/eLife.05563>.

http://www.eurekalert.org/pub_releases/2015-08/ind-btp080615.php

Bacteria that prevent type 1 diabetes

Micro-organisms can also protect us from certain diseases

Our bodies have ten times the amount of microbes than human cells. This set of bacteria is called microbiota. In some instances, bacteria known as pathogens can cause infectious diseases. However, these micro-organisms can also protect us from certain diseases. Researchers from Inserm, Paris Descartes University and the CNRS (French National Centre for Scientific Research), through collaboration with teams from China and Sweden, have recently shown how microbiota protects against the development of type 1 diabetes. This research is published in the *Immunity* journal, 4 August 2015.



A pancreatic islet of Langerhans is expressing the immunoregulator antimicrobial peptide CRAM (in red). The insulin-producing beta-cells are in green and the glucagon-producing alpha-cells are in blue. Julien Diana

To combat pathogens, the immune system has developed various mechanisms to detect, defend against and even destroy micro-organisms that are harmful to the body. This includes antimicrobial peptides and natural proteins that destroy pathogenic bacteria by disrupting their cellular membrane. Not only are they produced by immune cells, they are also produced by cells whose functions are not immune-related.

A research team coordinated by Julien Diana, an Inserm Research Fellow at Inserm Unit 1151 "Institut Necker-Enfant Malades" [Necker Institute for Sick Children] (Inserm/CNRS/Université Paris Descartes), is focussing on a category of antimicrobial peptides, i.e. cathelicidins. Apart from their protective function, these peptides have also exhibited immunoregulatory abilities against several autoimmune diseases.

As such, scientists hypothesise that cathelicidins may be involved in the control of type 1 diabetes, an autoimmune disease where certain cells in the immune system attack beta cells in the pancreas which secrete insulin.

Firstly, they observed that beta pancreatic cells in non-diseased mice produce cathelicidins and that, interestingly, this production is impaired in diabetic mice.

To test this hypothesis, they are injecting diabetic mice with cathelicidins where production is defective. "Injecting cathelicidins inhibits the development of pancreatic inflammation and, as such, suppresses the development of autoimmune disease in these mice" states Julien Diana.

Given that the production of cathelicidins is controlled by short-chain fatty acids produced by gut bacteria, Julien Diana's team are studying the possibility that this may be the cause of the cathelicidin deficiency associated with diabetes. Indeed, researchers have observed that diabetic mice have a lower level of short-chain fatty acids than that found in healthy mice.

By transferring part of the gut bacteria from healthy mice to diabetic mice, they are re-establishing a normal level of cathelicidin. Meanwhile, the transfer of micro-organisms reduces the occurrence of diabetes.

For the authors, "this research is further evidence of the undeniable role microbiota plays in autoimmune diseases, particularly in controlling the development of autoimmune diabetes". Preliminary data, as well as scientific literature, suggest that a similar mechanism may exist in humans, paving the way for new therapies against autoimmune diabetes.

Pancreatic beta-cells limit autoimmune diabetes via an immunoregulatory antimicrobial peptide expressed under the influence of the gut microbiota Immunity, 04 August 2015

Jia Sun¹, Laetitia Furio^{2,3}, Ramine Mecheri³, Anne M. van der Does⁴, Erik Lundberg⁴, Loredana Saveanu^{3,5}, Yongquan Chen¹, Peter van Endert^{3,5}, Birgitta Agerberth⁶, Julien Diana^{3,5}.

¹ State Key Laboratory of Food Science and Technology, Synergetic Innovation Center of Food Safety and Nutrition and School of Food Science and Technology, Jiangnan University, 1800 Lihu Avenue Wuxi, 214122 Jiangsu, P. R. China;

² French National Institute of Health and Medical Research (INSERM), Unit 1163, Institut Imagine, 24 Boulevard du Montparnasse, 75015 Paris, France;

³ Paris Descartes University, Sorbonne Paris Cité, 12 Rue de l'École de Médecine, 75006 Paris, France;

⁴ Department of Physiology and Pharmacology, Karolinska Institutet, SE-141 86 Stockholm, Sweden;

⁵ French National Institute of Health and Medical Research (INSERM), Unit 1151, Necker Institute for Sick Children (INEM), French National Scientific Research Centre, Unit 8253, 149 Rue de Sèvres, 75015 Paris, France;

⁶ Medical Microbial Pathogenesis Department of Laboratory Medicine, Clinical Microbiology, Karolinska Institutet Karolinska University Hospital, SE-141 86 Stockholm, Sweden.

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Paleo diet: Big brains needed carbs

The importance of dietary carbohydrate in human evolution

Understanding how and why we evolved such large brains is one of the most puzzling issues in the study of human evolution.

It is widely accepted that brain size increase is partly linked to changes in diet over the last 3 million years, and increases in meat consumption and the development of cooking have received particular attention from the scientific community.

In a new study published in The Quarterly Review of Biology, <http://www.jstor.org/stable/10.1086/682587>, Dr. Karen Hardy and her team bring together archaeological, anthropological, genetic, physiological and anatomical data to argue that carbohydrate consumption, particularly in the form of starch, was critical for the accelerated expansion of the human brain over the last million years, and coevolved both with copy number variation of the salivary amylase genes and controlled fire use for cooking.

With global increase in obesity and diet-related metabolic diseases, interest has intensified in ancestral or 'Palaeolithic' diets, not least because - to a first order of approximation - human physiology should be optimized for the nutritional profiles we have experienced during our evolution.

Up until now, there has been a heavy focus on the role of animal protein and cooking in the development of the human brain over the last 2 million years, and the importance of carbohydrate, particular in form of starch-rich plant foods, has been largely overlooked.

Hardy's team highlights the following observations to build a case for dietary carbohydrate being essential for the evolution of modern big-brained humans:

(1) The human brain uses up to 25% of the body's energy budget and up to 60% of blood glucose. While synthesis of glucose from other sources is possible, it is not the most efficient way, and these high glucose demands are unlikely to have been met on a low carbohydrate diet;

(2) Human pregnancy and lactation place additional demands on the body's glucose budget and low maternal blood glucose levels compromise the health of both the mother and her offspring;

(3) Starches would have been readily available to ancestral human populations in the form of tubers, as well as in seeds and some fruits and nuts;

(4) While raw starches are often only poorly digested in humans, when cooked they lose their crystalline structure and become far more easily digested;

(5) Salivary amylase genes are usually present in many copies (average ~6) in humans, but in only 2 copies in other primates. This increases the amount of salivary amylase produced and so increases the ability to digest starch. The exact date when salivary amylase genes multiplied remains uncertain, but genetic evidence suggests it was at some point in the last 1 million years.

Hardy proposes that after cooking became widespread, the co-evolution of cooking and higher copy number of the salivary amylase (and possibly pancreatic amylase) genes increased the availability of pre-formed dietary glucose to the brain and fetus, which in turn, permitted the acceleration in brain size increase which occurred from around 800,000 years ago onwards.

Eating meat may have kick-started the evolution of bigger brains, but cooked starchy foods together with more salivary amylase genes made us smarter still.

http://www.eurekalert.org/pub_releases/2015-08/aaft-qda080315.php

Gene deletions and duplications reveal our genetic storyline

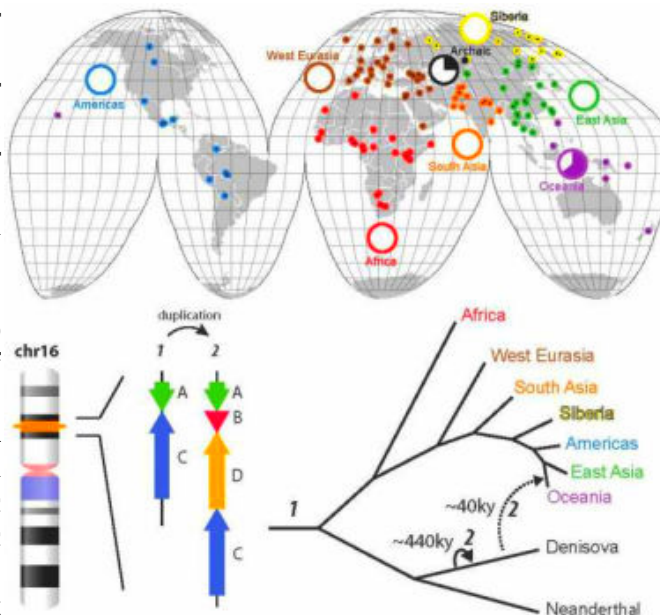
By looking closely at DNA variation across a vast number of populations, researchers now have a better idea of how selection affects the human genome around the globe.

This news release is [available in Japanese](#).

Copy number variation is the structural difference between genomes when large portions of DNA are duplicated or deleted. It can involve genomic regions containing multiple genes or important regulatory regions.

Because of this, it has been hypothesized that such changes are under selection (preferentially surviving, or being eliminated), but the forces eliminating or maintaining them within our genomes have not been well understood.

To gain a better understanding of these patterns globally, Peter Sudmant and colleagues analyzed copy number variation across 236 individual genomes from 125 human populations. Through this analysis, the researchers were able to identify patterns of ancestry as well as the maintenance of admixed genomes resulting from interbreeding of archaic hominids; Oceanic populations retain large duplications that originated in the Denisovan lineage.



Counterclockwise from the top: The geographic coordinates of populations sampled are indicated on a world map (colored dots). The pie charts show the continental population allele frequency of a single ~225 kbp duplication polymorphism found exclusively among Oceanic populations and an archaic Denisova. The ancestral structure of this duplication locus (1) and the Denisova duplication structure (2) are shown in relation to their position on chromosome 16. We estimate that the duplication emerged ~440 thousand years ago (kya) in the Denisova and then introgressed into ancestral Papuan populations ~40 kya. Peter H. Sudmant

As expected, Africans were more likely to show evidence of ancestral genome sequences compared to non-African populations, as the latter have experienced more population bottlenecks, resulting in lower levels of diversity. These bottleneck events for non-African populations have also resulted in fewer deletions compared to African populations.

Surprisingly, the results suggest that DNA deletions are more reflective of selection, whereas duplications better highlight genetic subpopulations. This variation in our genomes tells a fascinating story of human movement and colonization around the world, and the demographic and selective pressures humans have faced over generations.

Article #17: "Global diversity, population stratification, and selection of human copy number variation," by P.H. Sudmant; B.J. Nelson; F. Hormozdiari; N. Krumm; J. Huddleston; B.P. Coe; C. Baker; M. Bamshad; E.E. Eichle at University of Washington in Seattle, WA; S. Mallick; S. Nordenfelt; N. Patterson; D. Reich at Broad Institute of MIT and Harvard in Cambridge, MA S. Mallick; S. Nordenfelt; N. Patterson; D. Reich at Harvard Medical School in Boston, MA L.B. Jorde at University of Utah School of Medicine in Salt Lake City, UT. For a complete list of authors, see the manuscript.

http://www.eurekalert.org/pub_releases/2015-08/uonc-usp073115.php

UNC scientists pinpoint how a single genetic mutation causes autism

The research shows the precise cellular mechanisms that lead to the disorder and how an existing drug might help thousands of people with autism

CHAPEL HILL, NC - Last December, researchers identified more than 1,000 gene mutations in individuals with autism, but how these mutations increased risk for autism was unclear. Now, UNC School of Medicine researchers are the first to show how one of these mutations disables a molecular switch in one of these genes and causes autism.

Published today in the journal *Cell*, the research shows that an enzyme called UBE3A can be switched off when a phosphate molecule is tacked onto UBE3A. In neurons and during normal brain development, this switch can be turned off and on, leading to tight regulation of UBE3A. But a research team led by Mark Zylka, PhD, associate professor of cell biology and physiology, found that an autism-linked mutation destroys this regulatory switch. Destruction of the switch creates an enzyme that cannot be turned off. As a result, UBE3A becomes hyperactive and drives abnormal brain development and autism.

"Genetic studies are showing that there will be about 1,000 genes linked to autism. This means you could mutate any one of them and get the disorder. We found how one of these mutations works," said Zylka, senior author of the *Cell* paper and member of the UNC Neuroscience Center.

The work was done in human cell lines, as well as mouse models.

Because this one autism-linked UBE3A mutation was part of the Simons Simplex Collection - and Zylka previously had been funded through a Simons Foundation grant - he had access to the cells that were used to find this one mutation. When Jason Yi, PhD, a postdoctoral fellow in Zylka's lab, sequenced the genes from the cell samples - including cells from the child's parents - he found that the parents had no hyperactive UBE3A but the child did. The child's regulatory switch was broken, causing UBE3A to be perpetually switched on.

"When this child's mutation was introduced into an animal model, we saw all these dendritic spines form on the neurons," said, Zylka, who is also a member of the Carolina Institute for Developmental Disabilities. "We thought this was a big deal because too many dendritic spines have been linked to autism." Their findings thus pointed to hyperactivation of UBE3A as the likely cause of this child's autism.

It was previously thought that too much UBE3A might cause autism because duplication of the 15q chromosome region - which encompasses UBE3A and several other genes - is one of the most commonly seen genetic alterations in people with autism. This is called Dup15q Syndrome.

As part of their study, Zylka and Yi found that protein kinase A (PKA) is the enzyme that tacks the phosphate group onto UBE3A. This finding has therapeutic implications, particularly since drugs exist to control PKA.

"We think it may be possible to tamp down UBE3A in Dup15q patients to restore normal levels of enzyme activity in the brain," Zylka said. "In fact, we tested known compounds and showed that two of them substantially reduced UBE3A activity in neurons."

One of the drugs, rolipram, previously had been tested in clinical trials to treat depression but was discontinued due to side effects. One of the symptoms associated with Dup15q syndrome is sudden unexpected death in epilepsy. In light of these life-threatening seizures, Zylka pointed out that it may be worth examining whether lower doses of rolipram, or other drugs that increase PKA activity, provide some symptom relief in Dup15q individuals. "The benefits might outweigh the risks," he said. Future work with an animal model of Dup15q could be used to test this therapeutic approach.

While the bulk of this project was focused on autism, this project began when Zylka and Yi noticed that a large number of Angelman syndrome-linked mutations were clustered in the same chromosome region where the phosphate group was tacked onto UBE3A. Angelman syndrome is a rare neurological disorder characterized by developmental delay, seizures, balance problems, and lack of speech.

Zylka's team found that a number of the Angelman mutations disrupt the function or stability of UBE3A. These mutations would essentially eliminate the enzyme in people with Angelman syndrome. This discovery could have implications for diagnosing people with this rare and often misdiagnosed disorder.

Other co-authors included Ben Philpot, PhD, professor of cell biology and physiology, William Snider, MD, director of the UNC Neuroscience Center, and Klaus Hahn, PhD, the Ronald Thurman Distinguished Professor of Pharmacology. Janet Berrios, a graduate student at UNC, and Jason Newbern, PhD, assistant professor in the School of Life Sciences at Arizona State University, are also co-authors.

The National Institutes of Health, the Angelman Syndrome Foundation, The Foundation for Angelman Syndrome Therapeutics, and Autism Speaks funded this work.

http://www.eurekalert.org/pub_releases/2015-08/sflf-smh080615.php

Safe motorcycle helmets -- made of carrot fibers?

Detailed life cycle assessments for industrial products of the future

All over the world, research is being conducted into biodegradable and recyclable synthetics. However, fiber-reinforced components remain problematic - if glass or carbon fibers are used. Within the scope of an EU research project, the Scottish company Cellucomp Limited has now developed a method to obtain nanofibers from carrot waste. These fibers would be both cost-effective and biodegradable. However, is the method, which works in the lab, also marketable on a large scale?

An MPAS (multi-perspective application selection) method developed at Empa helps identify the industrial sectors where new materials might be useful from a technical and economical perspective. At the same time, MPAS also considers the ecological aspect of these new materials. The result for our example: Nanofibers made of carrot waste might be used in the production of motorcycle helmets or side walls for motorhomes in the future.

Three-step analysis

In order to clarify a new material's market potential, Empa researchers Fabiano Piccinno, Roland Hischier and Claudia Som proceed in three steps for the MPAS method. First of all, the field of possible applications is defined: Which applications come into question based on the technical properties and what categories can they be divided into? Can the new material replace an existing one? The second step concerns the technical feasibility and market potential: Can the material properties required be achieved with the technical process? Might the product quality vary from one production batch to the next? Can the lab process be upgraded to an industrial scale cost-effectively? Is the material more suited to the low-cost sector or expensive luxury goods? And finally: Does the product meet the legal standards and the customers' certification needs?

In the third step, the ecological aspect is eventually examined: Is this new material for the products identified really more environmentally friendly - once all the steps from product creation to recycling have been factored in? Which factors particularly need to be considered during production stage to manufacture the material in as environmentally friendly a way as possible?

Industrial production on a five-ton scale - calculated theoretically

The MPAS approach enables individual scenarios for a future production to be calculated with an extremely high degree of accuracy. In the case of the carrot waste nanofibers, for instance, it is crucial whether five tons of fresh carrots or only 209 kilograms of carrot waste (fiber waste from the juicing process) are used as the base material for their production.

The issue of whether the solvent is ultimately recycled or burned affects the production costs. And the energy balance depends on how the enzymes that loosen the fibers from the carrots are deactivated. In the lab, this takes place via heat; for production on an industrial level, the use of bleaching agents would be more cost-effective.

Conclusion: six possible applications for "carrot fibers"

For fiber production from carrot waste, the MPAS analysis identified six possible customer segments for the Scottish manufacturer Cellucomp that are worth taking a closer look at: Protective equipment and devices for recreational sport, special vehicles, furniture, luxury consumer goods and industrial manufacturing. The researchers listed the following examples: Motorcycle helmets and surfboards, side walls for motorhomes, dining tables, high-end loudspeaker boxes and product protection mats for marble-working businesses. Similarly detailed analyses can also be conducted for other renewable materials - before a lot of money is actually invested in production plants.

<http://bit.ly/1J4m3x0>

Microbes Deep under Seafloor Reflect Ancient Land Origins

Microbes 2,500 meters below the seafloor in Japan are most closely related to bacterial groups that thrive in forest soils on land, suggesting that they might be descendants of ones that survived when their terrestrial habitat was flooded 20 million years ago

By Cynthia Graber

[Download MP3](#)

Microbial organisms live in you, on you, in soil, in clouds and below the ocean floor. That last batch of single-celled critters is particularly hard to study. Not only are the samples difficult to obtain, but they can easily be contaminated with all that other microbial life once we dig them up.

So researchers had to take special precautions when they investigated microbial life in a coal bed deep below the seafloor near Japan. This material was once dry land, but got submerged some 20 million years ago.

The research team drilled nearly 2,500 meters below the seafloor and brought up samples. They carefully avoided contamination and evaluated only the inner portions of the samples, which were protected by the outer parts.

Analysis found life tenaciously holding on well under the ocean. A gram of rich garden soil can hold a billion bacteria—at 2,500 meters below the seafloor a gram of sediment might be home to just a single microbe. And those deeply buried organisms are quite different from microbes to be found just under the seafloor.

In that deep layer, the microbes are most closely related to bacterial groups that thrive in forest soils on land.

The scientists thus suggest that the deeply buried undersea microbes might be descendants of ones that survived when their terrestrial habitat became flooded. Again, that was 20 million years ago. The finding is in the journal *Science*. [F. Inagaki et al, Exploring deep microbial life in coal-bearing sediment down to ~2.5 kilometers below the ocean floor]

In a commentary in the same issue, Julie Huber of the Marine Biological Laboratory at Woods Hole says the fact that there's "a massive buried biosphere" has global importance "with sub-seafloor microbes playing a crucial role in carbon sequestration...and Earth's evolution, and likely encompassing staggering metabolic and genetic diversity." She adds, "We still have a long way to go in uncovering and understanding microbial life deep beneath the seafloor."

<http://bit.ly/1J4m3x0>

This Renaissance Painting of Fruit Holds a Modern-Day Science Lesson

Hint: it's in the watermelon

By [Helen Thompson](#)

Paintings can be a window to more than the outmoded dress and strange customs of the past — sometimes, they have modern-day science lessons to impart, too. That's the case with Giovanni Stanchi's 17th century still life of fruit, as Phil Edwards [points out](#) for *Vox* — just look for the watermelons.

Stanchi's work, painted between 1645 and 1672 (and now [up for auction](#) at Christie's), includes strange watermelons that look so foreign they could be from outer space in the bottom right corner. If watermelons looked like that in the Renaissance, then why do they look so different today? To delve into that question, Edwards spoke to James Neihuis, a horticulturist at the University of Wisconsin.

Watermelons had made their way to gardens in southern Europe by around 1600, writes Edwards, but they weren't domesticated by any means. For one thing, they were smaller, as most wild fruits are. They were also lighter than modern-day watermelons — the fleshy fruit surrounding the seeds is actually watermelon placenta, and it contained lower levels of lycopene, the protein that gives the fruit its red color.



Giovanni Stanchi (Rome c. 1645-1672). Oil on canvas, 38 5/8 x 52½ in. (Christie's Images Ltd.)

Over time, humans have selectively bred watermelons to grow larger and produce higher levels of lycopene, brightening their insides in the process.

Another striking thing about the Stanchi watermelons has little to do with domestication, though, [writes](#) Christopher Jobson for *This is Colossal*. The placenta appears divided and swirls around the seeds. That's a phenomenon called "[starring](#)," and it's a product of unfriendly growing conditions in which the plant has less access to pollen.

Still, Edwards notes, Stanchi's watermelons are a reminder that looking at art from hundreds of years ago can reveal a lot about humans have molded wild plants into modern crops. In their mere difference from the watermelons available in modern-day grocery stores, the fruit points to centuries of careful breeding — and a past in which fruit looked much different from today.

http://www.eurekalert.org/pub_releases/2015-08/uoc--psl_1080315.php

Pupil shape linked to animals' ecological niche

While the eyes may be a window into one's soul, new research led by scientists at the University of California, Berkeley, suggests that the pupils could also reveal whether one is a hunter or hunted.

Berkeley -- An analysis of 214 species of land animals shows that a creature's ecological niche is a strong predictor of pupil shape. Species with pupils that are vertical slits are more likely to be ambush predators that are active both day and night. In contrast, those with horizontally elongated pupils are extremely likely to

be plant-eating prey species with eyes on the sides of their heads. Circular pupils were linked to "active foragers," or animals that chase down their prey. The study, led by vision scientist Martin Banks, a UC Berkeley professor of optometry, in collaboration with the United Kingdom's Durham University, presents a new hypothesis as to why pupils are shaped and oriented the way they are. The findings will be published Friday, Aug. 7, in the journal *Science Advances*.

This current research builds upon the foundation set by the late Gordon Walls, a UC Berkeley professor of optometry who published "The Vertebrate Eye and Its Adaptive Radiation" in 1942. The classic text on eye physiology put forward the theory, generally accepted, that slit-shaped pupils allow for different musculature and a greater range in the amount of light entering the eye.

For example, the vertical slits of domestic cats and geckos undergo a 135- and 300-fold change in area between constricted and dilated states, while humans' circular pupils undergo a mere 15-fold change.

Why not diagonal slits?

"For species that are active both night and day, like domestic cats, slit pupils provide the dynamic range needed to help them see in dim light yet not get blinded by the midday sun," said Banks. "However, this hypothesis does not explain why slits are either vertical or horizontal. Why don't we see diagonal slits? This study is the first attempt to explain why orientation matters."

To explain why horizontally elongated pupils, with few exceptions (are there any explanations for the exceptions?), corresponded to grazing prey animals such as sheep, deer and horses, the researchers turned to computer models to study the effects of different pupil shapes.

They found that the horizontal pupils expanded the effective field of view. When stretched horizontally, the pupils are aligned with the ground, getting more light in from the front, back and sides. The orientation also helps limit the amount of dazzling light from the sun above so the animal can see the ground better, the researchers said. "The first key visual requirement for these animals is to detect approaching predators, which usually come from the ground, so they need to see panoramically on the ground with minimal blind spots," said Banks. "The second critical requirement is that once they do detect a predator, they need to see where they are running. They have to see well enough out of the corner of their eye to run quickly and jump over things."

But what happens to this orientation when the animal lowers its head to graze? If the pupil follows the pitch of the head, they would become more vertical and the theory falters. "To check this out, I spent hours at the Oakland Zoo, often surrounded by school kids on field trips, to observe the different animals," said Banks. "Sure enough, when goats, antelope and other grazing prey animals put

their head down to eat, their eyes rotated to maintain the pupils' horizontal alignment with the ground."

On the other side of the Atlantic, study co-author Gordon Love, a professor of physics at Durham University, found this same pattern when observing sheep and horses at nearby farms. Grazing animals' eyes can rotate by 50 degrees or more in each eye, a range 10 times greater than human eyes, the researchers said.

How ambush predators focus when catching prey

For ambush predators with vertical-slit pupils, the authors noted the importance of accurately gauging the distance animals would need to pounce on their prey. Researchers identified three cues generally used to gauge distance: stereopsis, or binocular disparity; motion parallax, in which closer objects move farther and faster across our field of vision; and blur, in which objects at different distances are out of focus.

The researchers ruled out motion parallax as a factor since using that cue would require head movement that could reveal the predator's position. The remaining two cues, binocular disparity and blur, work together with vertically elongated pupils and front-facing eyes, the researchers said.

Binocular vision works better at judging differences when contours are vertical and objects are at a distance, while blur comes into play for horizontal contours and near-field targets. Vertical-slit pupils maximize both cues, the researchers said. Vertical pupils are not equally distributed among ambush predators, however.

"A surprising thing we noticed from this study is that the slit pupils were linked to predators that were close to the ground," said William Sprague, a postdoctoral researcher in Banks' lab. "So domestic cats have vertical slits, but bigger cats, like tigers and lions, don't. Their pupils are round, like humans and dogs."

Among the 65 frontal-eyed, ambush predators in this study, 44 had vertical pupils, and 82 percent of them had shoulder heights that were less than 42 centimeters (16.5 inches). Vertical pupils appear to maximize the ability of small animals to judge distances of prey.

The authors explained this by calculating that depth-of-field cues based upon blur are more effective for estimating distances for short animals than tall ones. "We are learning all the time just how remarkable the eye and vision are," said Love. "This work is another piece in the jigsaw puzzle of understanding how eyes work." The authors noted that this research focused on terrestrial species. They expect to examine associations of aquatic, aerial and arboreal life on eye position and pupil shape in future studies.

Other co-authors of this study are Jürgen Schmoll and Jared Parnell at Durham University.

The National Institutes of Health and the Engineering and Physical Sciences Research Council helped support this research.

http://www.eurekalert.org/pub_releases/2015-08/uoca-sfi080615.php

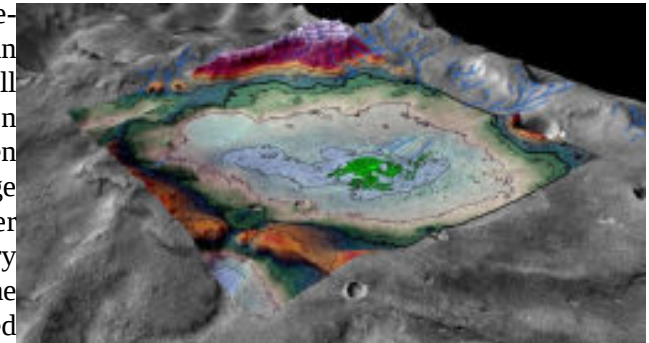
Salt flat indicates some of the last vestiges of Martian surface water

Ancient lake may represent last potentially habitable surface water to exist on Mars

Mars turned cold and dry long ago, but researchers at the University of Colorado Boulder have discovered evidence of an ancient lake that likely represents some of the last potentially habitable surface water ever to exist on the Red Planet.

The study, published Thursday in the journal *Geology*, examined an 18-square-mile chloride salt deposit (roughly the size of the city of Boulder) in the planet's Meridiani region near the Mars Opportunity rover's landing site. As seen on Earth in locations such as Utah's Bonneville Salt Flats, large-scale salt deposits are considered to be evidence of evaporated bodies of water.

Digital terrain mapping and mineralogical analysis of the features surrounding the deposit indicate that this one-time lakebed is no older than 3.6 billion years old, well after the time period when Mars is thought to have been warm enough to sustain large amounts of surface water planet-wide. Planetary scientists believe that the solar system formed approximately 4.6 billion years ago.



This is a perspective rendering of the Martian chloride deposit. LASP / Brian Hynek

"This was a long-lived lake, and we were able to put a very good time boundary on its maximum age," said Brian Hynek, a research associate at the Laboratory for Atmospheric and Space Physics (LASP) at CU-Boulder and lead author of the study. "We can be pretty certain that this is one of the last instances of a sizeable lake on Mars."

Based on the extent and thickness of the salt, the researchers estimate that the lake was only about 8 percent as salty as the Earth's oceans and therefore may have been hospitable to microbial life.

"By salinity alone, it certainly seems as though this lake would have been habitable throughout much of its existence," said Hynek, who is also an associate professor in the Department of Geological Sciences at CU-Boulder and director of

the CU Center for Astrobiology. He noted, however, that other factors such as acidity levels were not included in the scope of the study.

Mikki Osterloo and Kathryn Kierein-Young, both research associates at the Laboratory for Atmospheric and Space Physics (LASP) at CU-Boulder, co-authored the study.

The University of Alaska Geophysical Institute's Remote Sensing Laboratory and the University of Arizona's High Resolution Imaging Science Experiment (HiRISE) team provided assistance with digital terrain mapping and data processing. The NASA-Mars Data Analysis Program provided funding for the research.

<http://www.medscape.com/viewarticle/848959>

HPV Vaccines: Who, What, and When?

Updated Guidance

Sandra Adamson Fryhofer, MD

Available HPV Vaccine Products

Hello, I'm Dr Sandra Fryhofer. Welcome to Medicine Matters. The topic: HPV vaccination, the transition to HPV9, and gender differences in additional cancer protection. HPV—the human papillomavirus—is linked to cancer, including cervical, vulvar, and vaginal cancer in females; penile cancer in males; and anal and oropharyngeal cancer in both males and in females.

Three HPV vaccines are now available:

- **HPV2 (Cervarix®), US Food and Drug Administration (FDA)-approved in 2009;**
- **HPV4 (Gardasil®), FDA-approved in 2006; and**
- **HPV9 (Gardasil® 9), FDA approved in December 2014.**

What Do They Protect Against?

All three vaccines provide cancer protection against HPV types 16 and 18, the types that cause most (64%) of all HPV cancers. These HPV types are to blame for an estimated 21,300 cases of cancer each year. Types 16 and 18 are linked to 63% of all HPV-related cancers in males. In females, HPV 16 and 18 cause 65% of all HPV-related cancers, as well as 66% of all cervical cancers.^[1] The HPV4 and HPV9 vaccines also protect against types 6 and 11—the types that cause 90% of all anogenital warts.^[1] The newest HPV vaccine (HPV9) includes additional protection from five cancer-causing HPV strains: 31, 33, 45, 52, and 58. The HPV9 vaccine is more expensive than its HPV4 sibling.

But how much extra cancer protection do patients receive from the additional coverage and cost? Overall, about 10% of HPV-related cancers (about 3400 cancer cases each year) are linked to the five additional strains.

Are gender differences in the additional cancer protection conferred? Most of the added protection from covering the five additional types is for females. For males, HPV9 provides only 4% additional cancer protection. For females, the additional strains cause 14% of HPV cancers overall, including 15% of cervical cancers and 25% of cervical precancers.^[1]

Who, What, and When?

Here is the latest Advisory Committee on Immunization Practices (ACIP) recommendation for HPV vaccination, published in the March 27, 2015, *Morbidity and Mortality Weekly Report*^[2]:

- **Three HPV vaccine doses are recommended routinely starting at age 11 or 12 years, but vaccination can begin as early as age 9.**
- **HPV vaccination is recommended through age 26 years for all females and through age 21 years for all males.**
- **Vaccination through age 26 years is also recommended for immunocompromised males, including those with HIV, and for men who have sex with men.**
- **HPV9 is one of three HPV vaccines that can be used for routine vaccination:**
 - HPV2 is licensed only for females.
 - HPV4 and HPV9 are licensed for both males and females.
 - **Three doses of vaccine are needed. Any of the three vaccines can be used to start, continue, or complete the series for females. Either HPV4 or HPV9 should be used to start, complete, or continue the series in males.**

What to Do With Leftover HPV4 Vaccine?

Let's now try to clear up some confusion about HPV9. The package insert says that HPV9 is licensed for females aged 9-26 years and for males aged 9-15 years.^[3] ACIP reviewed additional data on HPV9 in males aged 16-26 years in making its new HPV9 recommendation. It's fine to use HPV9 in males through age 26 years. The company has submitted these data to the FDA with a request to expand the age indication of HPV9 for males.

The introduction of HPV9 has created some programmatic issues. Many practitioners still have HPV4 vaccine on hand. Merck says it has no plans to exchange remaining HPV4 vaccine for HPV9. It plans to have quadrivalent vaccine available for at least 6 months after FDA approval (when and if that occurs) of the expanded HPV9 age indication of 16-26 years for males.

The incremental cancer protection from HPV9 for males is small. For this reason, one option is to give any remaining HPV4 vaccine to male patients. Although this is not an official ACIP recommendation, it was suggested as a strategy for Vaccines for Children (VFC) awardees at the June 2015 ACIP meeting.

You May Also Be Wondering About...

The ACIP website will soon be posting some [official guidance about additional HPV9 vaccine](#) doses and how to complete an HPV vaccination series started with another HPV vaccine. Here are some highlights reviewed at the June ACIP meeting^[4]:

- **There is no ACIP recommendation for routine additional HPV9 vaccination for anyone who has already completed the bivalent or quadrivalent vaccination series.**

- Available studies show no serious safety concerns with giving HPV9 to those that have completed a three-dose HPV vaccine series. However, they did have higher rates of injection site swelling and redness.
- The guidance also emphasizes gender differences in protection. The benefit of protection against the five additional strains in HPV9 is mostly limited to females, in protecting from cervical cancers and cervical precancers.
- The guidance also mentions a study (found in the HPV9 package insert) in which females who had already completed a three-dose HPV4 series were (starting a year later) given three doses of HPV9 vaccine. These patients did make antibodies to the five additional vaccine types, but the titers were lower; they were 25%-63% of the titers found in patients receiving HPV9 without previous HPV vaccination. The clinical significance of these lower antibody titers is not known.

There is a trial under way looking at the effectiveness of two HPV9 doses separated by 6 or 12 months in HPV-vaccine-naïve individuals. The results of the study may be available in late 2015.

For Medicine Matters, I'm Dr Sandra Fryhofer.

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<http://bit.ly/1MoiTqI>

Fish Slime Inspires New Eco-Sunscreen Ingredient

Researchers have developed a new ecofriendly sunscreen molecule that protects against both UV-A and UV-B rays, and could also be used to create more durable paints and plastics. Christopher Intagliata reports

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Anyone who's gone snorkeling at a [coral reef](#) lately may have been [discouraged from slathering on too much sunscreen](#). In some places, the nonbiodegradable skin protection [is actually banned](#). That's because researchers reckon that some

[four to six thousand tons](#) of [sunscreen](#) float off the skin of snorkelers every year, enveloping corals in a cloud of chemicals—chemicals that can [sicken or even bleach the coral](#).

But a more ecofriendly way of saving our skin might be to copy *nature's* tricks. Algae and cyanobacteria produce sunlight-absorbing compounds. So do reef-dwelling fish, in the protective slime on their bodies. Researchers isolated those molecules, called mycosporines, which absorb both UV-A and UV-B rays.

Mycosporines have actually been used before in a few SPF products, but in a form that can both penetrate our skin, and easily wash off.

So the biochemists attached the mycosporines to chitosan, a polymer derived from the shells of shrimp and crabs. This hybrid package, they say, is a more effective sunscreen, with constituents too big to pass into the skin, and it's more resistant to washing off. It's also hypoallergenic, and did not affect cell development, in in-vitro tests. The findings appear in the journal *Applied Materials & Interfaces*. [Susana C. M. Fernandes et al, [Exploiting Mycosporines as Natural Molecular Sunscreens for the Fabrication of UV-Absorbing Green Materials](#)]

The researchers say that besides providing a superior sunscreen, this material could also lead to more durable paints and plastics—think lawn chairs, and other outdoor items that take a beating from light and heat. And to be clear—this stuff is not being bottled just yet. But it could be soon, they say. Which could help protect the environment, in addition to saving our skin.

<http://www.bbc.com/news/health-33802686>

The vet will see you now...

How does the care and compassion shown to animals when they are sick compare to the treatment patients receive in the NHS?

"I'm really impressed with the care she gets," says GP Dr Graham Easton of his dog, Molly.

There are, of course, a number of important differences between the care humans and animals receive. In the UK, veterinary care is not free at the point of delivery whereas in the NHS, every human patient is treated equally according to their need and not their bank balance.

Arguably, the health system is under severe strain as a result. Dealing with complex human beings is very different to dealing with sick animals, but it has been suggested that lessons could be learned from watching how vets organise and deliver care and, particularly, how animals are looked after at the end of their lives.

In a vets' practice in Chipping Norton, there is a separate waiting area for dogs and cats. According to head vet, Martin Whitehead, they have made a point of thinking about the experience of their patients.

"It's not nice for a cat to be seated next to a big bulky dog," he says. There's a big airy treatment room and a diagnostics lab - complete with an X-ray and ultrasound scanner for looking at the abdomen and the heart - and it's all under one roof.

When a blood sample from Biggles the boxer, who has cancer and is off his food, is taken for testing in the in-house lab, the results come back very quickly.

GP Graham Easton is envious. saying: "It would be lovely to get blood results back that quickly. Not many GPs' surgeries can offer that kind of service."

Vets are true generalists, in every sense of the term. They can take care of pets with cancer and carry out surgery on a cruciate ligament injury in a dog, for example, without the need for referral to a specialist or the mention of a long waiting list.

But it all comes down to whether the owner can afford it. The private care system vets operate under means owners have to pay for the care their pets receive, or take out pet insurance to help pay for it. This is likely to make owners think before they attend, which helps make the system more efficient.

However, a lack of evidence in veterinary medicine means vets have little research to back up their choice of treatment - and this could lead to unnecessary operations, Mr Whitehead suggests. "It would be lovely to have a big database of evidence to rely on," he says.

In contrast, human medicine is well-researched and very evidence-based. The guidelines are written down and recommended. On the whole, doctors know if people are going to benefit from a particular treatment or not. They can also communicate with a human patient and discuss the best options for his or her treatment. But the biggest challenges for the NHS are in how the systems work.

A common complaint is that it's difficult to get an on-the-day GP appointment without seeing a different GP from the previous time, thereby losing something very precious to patients - "continuity of care". So couldn't doctors take a leaf out of vets' books and work together more closely to make their services more efficient?

This is starting to happen in the NHS with GPs' surgeries now delivering a wider range of community services which are tailored to the needs of its patients.

While "integrated care" isn't appropriate or possible everywhere in the NHS, it is bringing some groups of health professionals together to improve patient care.

Hugh Alderwick, a healthy policy advisor at the Kings Fund - an independent charity which works to improve health care in England - says it's about "seeing care through the eyes of the patient".

Dr Easton agrees, saying: "Wouldn't it be lovely to see the same doctor every time? People really value it, particularly when things get difficult." He says

understanding, compassion and sensitivity are noticeable in the way vets treat animals.

Yet these are qualities that patients often say are missing in the NHS. Lengthy waits, poor care and a lack of communication are accusations frequently directed at the health service. Without doubt there are many compassionate staff in the NHS, but perhaps when the system is overstretched and stressed, there is less time and space for health professionals to show it. Is it possible that at the end of life animals get better care too?

Prof Bob Michell, a past president of the Royal College of Veterinary Surgeons, thinks so. He lost his wife to pancreatic cancer and saw her suffer greatly during her last few weeks, despite excellent palliative care. In contrast, he says, their beloved Labrador was "eased into sleep to the sound of birds singing" and the contrast has always haunted him.

He believes humans should be able to choose how and when they die. "Few things are more important in patient choice than the circumstances of one's death. Why set a higher value on human life and then let people suffer?"

However Prof Irene Higginson, a palliative care consultant at King's College London, says the comparison with animals is a dangerous one because it is always the owner who decides on the time of death, rather than the animal.

She is concerned that some people would be rushed towards euthanasia because of poor care and a lack of hospice spaces, and because they fear being a burden on their family. "The emphasis should be on improving their quality of life. A lot can be done to look after the whole person and their family," she says.

Dr Easton is fiercely proud of the NHS. It may be stretched to the limit, he says, but it does a challenging job on complex human beings.

Yet, perhaps medics could learn from the customer focus that comes from private care - the ease of access, compassion and sensitive communication. "Medics and vets spending time in each other's worlds would be no bad thing," he says.

http://www.eurekalert.org/pub_releases/2015-08/tl-tld080615.php

The Lancet Diabetes & Endocrinology: Universal iodine supplementation during pregnancy could offer huge cost savings

Giving all pregnant women iodine supplements could result in huge cost savings for health care systems and society

Giving all pregnant women iodine supplements, even in mildly iodine deficient countries like the UK, could result in huge cost savings for health care systems and society, according to new modelling research published in The Lancet Diabetes & Endocrinology journal.

The new estimates suggest that introducing iodine supplementation in pregnancy in the UK could save the National Health Service (NHS) around £200 per expectant mother and provide monetary benefits to society of around £4500 per child from increased lifetime earnings and lower public sector costs. With around 1.9 billion people and 241 million school-age children (aged 6-12 years) living in the 32 countries that have iodine deficiency, the authors conclude that the benefits of universal iodine supplementation during pregnancy could be substantial.

"Iodine deficiency in pregnancy remains the leading cause of preventable retardation worldwide. Even mild iodine deficiency during pregnancy is associated with children with lower IQs," explains Kate Jolly, a co-author and Professor of Public Health at the University of Birmingham in the UK. "It's time for all women living in iodine deficient countries without universal supplementation of iodine, who are pregnant, breastfeeding, or planning a pregnancy to be advised to take a daily supplement containing iodine." [1]

Iodine is not made naturally in the body and must be consumed by eating foods like dairy and seafood or supplements. Severe iodine deficiency during pregnancy can cause substantial mental impairment and delayed development in children, resulting in a lower IQ and consequently lower educational attainment and earning potential. International health organisations like WHO and the European Food Safety Authority recommend that pregnant and breastfeeding women take daily iodine supplements. However, no recommendation for iodine supplementation has been issued to pregnant women in the UK, even though mild iodine deficiency has been reported to be widespread.

As a randomised trial might not be approved because of ethical concerns in the untreated group, a team of researchers from the University of Birmingham did a modelling study to examine the cost-effectiveness of iodine supplementation versus no supplementation for pregnant women in the UK. Using data from a systematic review of published studies and expert opinion they modelled both the direct health service savings and monetary benefits to society (lifetime earnings) in terms of gains from an additional IQ point in the children.

By converting the effects of iodine supplementation in pregnancy on developing brains into IQ points, the authors estimate that the benefits equate to 1.22 IQ points per child, with monetary benefits of around £199 per expectant mother for the NHS, and £4476 per pregnancy for society (table 2).

According to the authors, "As food fortification alone may not be enough to achieve iodine sufficiency for pregnant women, our results strengthen the case for universal iodine supplementation of all women before and during pregnancy and whilst breastfeeding in mild-to-moderate iodine deficient countries."

http://www.eurekalert.org/pub_releases/2015-08/uoea-cmc080715.php

Common medications could delay brain injury recovery

Anticholinergics could delay the recovery of brain injury patients

Drugs used to treat common complaints could delay the recovery of brain injury patients according to research led by University of East Anglia (UEA) scientists working with other UK universities including Aston and the NHS, published today in Brain Injury.

Prescribed for up to 50 per cent of older people, medications with anticholinergic properties are used to treat a broad range of common conditions including bladder problems, depression and insomnia.

Anticholinergics are already known to have side effects such as temporary cognitive impairment, dizziness and confusion. But their effects on people with pre-existing brain and spinal injuries have not been investigated until now.

Medications with anti-cholinergic properties are often used on neuro-rehabilitation units frequently to manage symptoms from urinary incontinence to pain.

The study of 52 patients with acquired brain or spinal injury at a neuro-rehabilitation unit showed that the average length of stay was longer in patients with a higher level of anticholinergic drugs in their system, known as the anticholinergic drug burden, or ACB.

Results showed that the change in ACB correlated directly to the length of hospital stay. A higher ACB score on discharge, compared with on admission, was associated with a longer stay in hospital and a lower ACB on discharge saw on average a shorter stay. The team cautioned however that as an observational study, cause-and-effect relationship cannot be implied.

Dr Chris Fox, Professor of Clinical Psychiatry at the Norwich Medical School at UEA and lead author on the paper, said: "The findings suggest there may be a statistically significant relationship between ACB score and length of stay in a neuro-rehabilitation unit following traumatic brain or spinal cord injury".

He added: "This pilot study demonstrates the need for larger studies to confirm the results and need for further investigation into what long-term effects these common medications are having on the recovery of these patients."

"While medications with ACB are often needed to treat common complications of brain or spinal cord injuries, cognitive impairment due to the medication may adversely affect a patient's ability to engage in the rehabilitation process, potentially increasing their length of stay in hospital."

Length of patient stay is used a performance indicator for hospitals, with financial incentives in place for units to discharge patients as soon as is safe.

Dr Ian Maidment, Senior Lecturer in Clinical Pharmacy at Aston University said: "This work adds to the evidence that anticholinergics should be avoided in a wide-range of populations, when possible. Regular medication review by a nurse, doctor or pharmacist may be a way of ensuring that medicines with anticholinergic effects are used appropriately."

Prof Fox said: "Identifying factors which might adversely affect the length of a patient's stay can have important financial as well as quality of life implications. So the findings of this study could be directly useful to current health care settings if they can reduce the time patients spend in rehabilitation units, improving wider efficiency of care."

'Does anticholinergics drug burden relate to global neuro-disability outcome measures and length of hospital stay?' is published in the journal *Brain Injury* on Monday 10 August 2015.

http://www.eurekalert.org/pub_releases/2015-08/uos-pdf080715.php

Promising drug for Parkinson's disease: Study supports fast track to clinical trials

Marked rescue effect of the drug UDCA on cell batteries (mitochondria) in Parkinson's disease patient tissue

First study to demonstrate beneficial effects of UDCA on the nerve cells affected in Parkinson's disease in a genetic animal model of Parkinson's disease

UDCA is already approved for use in human liver disease

Results of the study support fast track of UDCA to clinical trials and could save years of research and hundreds of millions of pounds

A drug which has already been in use for decades to treat liver disease could be an effective treatment to slow down progression of Parkinson's disease, scientists from the University of Sheffield have discovered.

The pioneering research led by academics from the Sheffield Institute of Translational Neuroscience (SITraN), in collaboration with scientists from the University of York, supports the fast-tracking of the drug ursodeoxycholic acid (UDCA) for a clinical trial in Parkinson's patients.

Dr Heather Mortiboys, Parkinson's UK Senior Research Fellow from the University of Sheffield, explained: "We demonstrated the beneficial effects of UDCA in the tissue of LRRK2 carriers with Parkinson's disease as well as currently asymptomatic LRRK2 carriers. In both cases, UDCA improved mitochondrial function as demonstrated by the increase in oxygen consumption and cellular energy levels."

Oliver Bandmann, Professor of Movement Disorders Neurology at the University of Sheffield and Honorary Consultant Neurologist at Sheffield Teaching Hospitals NHS Foundation Trust, added: "Whilst we have been looking at Parkinson's

patients who carry the LRRK2 mutation, mitochondrial defects are also present in other inherited and sporadic forms of Parkinson's, where we do not know the causes yet. Our hope is therefore, that UDCA might be beneficial for other types of Parkinson's disease and might also show benefits in other neurodegenerative diseases."

The research is also the first to demonstrate beneficial effects of UDCA on dopaminergic neurons, the nerve cells affected in Parkinson's disease, in a fly model of Parkinson's disease which carries the same genetic change as some patients with the condition.

The study published in the journal *Neurology* is funded by Parkinson's UK, the Wellcome Trust and the Norwegian Parkinson Foundation.

A mutation in the LRRK2 gene is the single most common inherited cause of Parkinson's disease. However, the precise mechanism that leads to Parkinson's is still unclear.

Defects in mitochondria, and as a consequence reduced energy levels, are a factor in a number of diseases that affect the nervous system including Parkinson's and Motor Neuron Disease. Nerve cells have a particularly high energy demands, therefore defects in the cell's energy generators will crucially affect their survival. Professor Bandmann added: "Following on from the promising results of our in vitro drug screen, we were keen to further investigate and confirm the potential of UDCA in vivo - in a living organism.

"UDCA has been in clinical use for decades and thus could be advanced to the clinic rapidly if it proves beneficial in clinical trials."

Collaborators Rebecca Furnston, White Rose PhD student, and Dr Chris Elliott, from the University of York's Department of Biology, demonstrated the beneficial effects of UDCA in vivo using the fruit fly (*Drosophila melanogaster*). In fruit flies, the mitochondrial defects caused by the LRRK2 mutation to dopaminergic neurons can be monitored through the progressive loss of visual function. Flies carrying the mutation maintained their visual response when fed with UDCA.

Dr Elliott said: "The treatment of fruit flies carrying the faulty LRRK2 gene with UDCA showed a profound rescue of dopaminergic signalling. Feeding the flies with UDCA partway through their life slows the rate at which the fly brain then degenerates. Thus, mitochondrial rescue agents may be a promising novel strategy for disease-modifying therapy in LRRK2-related Parkinson's."

Dr Arthur Roach, Director of Research and Development at Parkinson's UK, which part-funded the study, said: "There is a tremendous need for new treatments that can slow or stop Parkinson's."

"Because of this urgency, the testing of drugs like UCDA, which are already approved for other uses, is extremely valuable. It can save years, and hundreds of millions of pounds.

"It's particularly encouraging in this study that even at relatively low concentrations the liver drug still had an effect on Parkinson's cells grown in the lab.

"This type of cutting-edge research is the best hope of finding better treatments for people with Parkinson's in years, not decades."

Reference: Mortiboys, H., Furnston, R., Bronstad, G., Aasly, J., Elliott, C., Bandmann, O. (2015) UDCA exerts beneficial effect on mitochondrial dysfunction in LRRK2G2019S carriers and in vivo. Neurology 85:1-7

The Sheffield Institute for Translational Neuroscience (SITraN) SITraN is a world-leading research centre purpose-built and dedicated to research into motor neuron disease (MND/ALS) and related neurodegenerative diseases including Parkinson's disease, Alzheimer's disease, and stroke. The state-of-the art research facility was opened in 2010 by HM The Queen and uniquely allows the multidisciplinary collaboration of clinicians, scientists and health professionals to develop new treatments for the benefit of patients. To find out more visit <http://www.sheffield.ac.uk/sitran/>