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Our hearts beat with unexpected electrical help from immune cells

Macrophages known for gobbling germs may be key to healthy—and unhealthy—rhythms.

Beth Mole - 4/21/2017, 4:06 AM

Having a regular or irregular heartbeat may come down to moonlighting immune cells that surprisingly help power blood-pumping pulses, a new study in Cell suggests.

In a series of experiments, Harvard researchers caught immune cells hanging around and helping heart cells conduct electricity for their rhythmic beats. The immune cells, called macrophages, are best known for surveilling the body and devouring invading germs and debris. But in the heart, they snuggled up to heart cells and formed pores through which electrical current could pulse through the organ, allowing for synchronous heart muscle contractions that pump blood. The macrophages also helped neighboring heart cells recharge between pulses.

In genetically engineered mice, a lack of macrophages in the heart led to irregular heartbeats that, in humans, would warrant implanting a pacemaker, the researchers found. In all, the finding suggests that macrophages are unexpectedly key to normal heart functioning—and could be behind some mysterious heart problems.

Further research, the authors write, could help physiologists better understand how the heart works and develop new types of therapies.

"This work opens up a completely new view on electrophysiology; now, we have a new cell type on the map that is involved in conduction," lead author Matthias Nahrendorf, a systems biologist at Harvard, said in a press release.

The researchers got the idea to look into macrophages after recent research caught them taking up second jobs in organs. Some macrophages rove widely through the body, gobbling garbage and germs as they go, but others patrol specific organs and tissues. Those stationary guards seem to do their best at blending in and helping out. For instance, in the liver and spleen, macrophages take on the task of iron recycling.

The researchers peeked into the hearts of mice and autopsied humans and found macrophages mingling with heart cells. There, the macrophages took on heart-specific cell shapes, allowing them to form electrical pores that connected them with their neighbors. In experiments, the macrophages unexpectedly displayed fluctuating electrical charges, which just happened to be synchronized with those of the heart cells.

With the mouse data, the researchers speculate that macrophages could play a role in conduction abnormalities, such as atrial fibrillation and certain arrhythmias. Further study on these cells may steer researchers to new therapies, and it may explain why anti-inflammatory drugs can sometimes help with heart disease.

Cell, 2017. DOI:10.1016/j.cell.2017.03.050 (About DOIs).

http://bbc.in/2oPdZtP

Global plan to wipe out hepatitis

Countries must work together to wipe out viral hepatitis - a disease that is killing almost as many people globally as HIV and TB, says the World Health Organization (WHO).

The death toll in 2015 was 1.34 million people, a new report reveals.

An estimated 325 million people are living with chronic hepatitis caused by B or C virus infection. Hepatitis vaccines and medicines exist, but they are not yet reaching everyone in need. This is partly because infections are not always identified - just 9% of all hepatitis B infections and 20% of all hepatitis C infections were diagnosed in 2015. As a result, millions of people are at risk of a slow progression to chronic liver disease, cancer and death, says the WHO.

Viral hepatitis

Viral hepatitis refers to five different forms of virus, known as A, B, C, D, E. Some (hepatitis B, C and D) can be spread through contact with

infected bodily fluids, including blood, while others (hepatitis A and hepatitis E) are spread through contaminated food or water.

In some parts of the world, including regions within Africa and the Western Pacific, hepatitis B and C infections are all too common.

Hepatitis B infection requires lifelong treatment - the WHO currently recommends the medicine tenofovir, already widely used in HIV treatment - but hepatitis C can be cured with a course of antiviral drugs.

The WHO says some countries are taking successful steps to scale up hepatitis services:

China achieved high coverage (96%) for the timely birth dose of HBV vaccines, and reached the hepatitis B control goal of less than 1% prevalence in children under the age of five in 2015

Mongolia improved uptake of hepatitis treatment by including hepatitis B and C medicines in its National Health Insurance scheme, which covers 98% of its population

In Egypt, market-price competition has reduced the cost of a three-month cure for hepatitis C, from $900 (£700) in 2015 to less than $200 in 2016

Dr Gottfried Hirnschall, from the WHO, said: "We are still at an early stage of the viral hepatitis response, but the way forward looks promising. "More countries are making hepatitis services available for people in need - a diagnostic test costs less than $1, and the cure for hepatitis C can be below $200.

"But the data clearly highlight the urgency with which we must address the remaining gaps in testing and treatment."

Raquel Peck, from the World Hepatitis Alliance, said: "Today, 325 million men, women and children are living with a cancer-causing illness, despite the availability of preventative vaccines for hepatitis B and curative treatments for hepatitis C.

"We need to use this report to advocate for a public health approach, so that testing and treatment are rolled out at the scale necessary to ensure that every person has the opportunity to live a healthy life."

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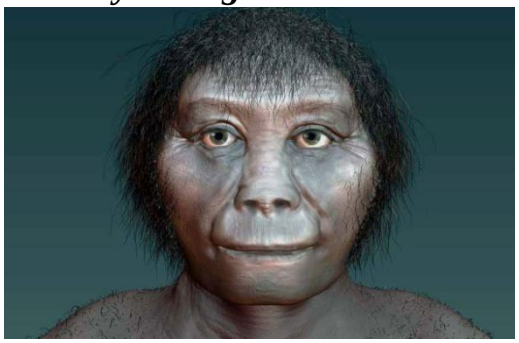
Mystery human hobbit ancestor may have been first out of Africa

The tiny Indonesian hominin may have descended from a species that left Africa 2 million years ago

By Alice Klein



Katrina Kenny

The identity of the mysterious *Homo floresiensis*, aka the hobbit, has once again been turned on its head. New research suggests the tiny hominin evolved from an unknown ancestor that was the first to ever venture out of Africa.

Remains of the extinct species were first discovered on the island of Flores in Indonesia just over a decade ago, but there is still fierce debate about where they came from.

The dominant idea has been that *H. floresiensis* was descended from the larger *Homo erectus*, an extinct human species that once occupied Asia. Proponents believe ancestors of *H. erectus* were the first humans to stray out of Africa about 1.8 million years ago.

The theory is that after members of the big-bodied group reached Flores, they gradually shrunk to just 1 metre tall because of the scarce island resources.

Another possibility is that the hobbits were simply short members of our own species – *Homo sapiens*. The miniature size of the one skull that has been uncovered could be the result of Down syndrome.

Now, the most comprehensive analysis yet suggests the hobbits were, in fact, descended from a mystery ancestor that lived in Africa over 2 million years ago. Some members of this ancestral group remained in Africa and evolved into *Homo habilis* – the first makers of stone tools. The others moved out of Africa about 2 million years ago – before *H. erectus* did – and arrived in Flores at least 700,000 years ago.

First to Flores

"As this ancestor spread through south and south-east Asia and then finally onto Flores, it would have gradually changed, finally becoming *H. floresiensis*," says Colin Groves at the Australian National University, who co-authored the study.

His team constructed the hobbit's family tree by carefully comparing skull, jaw, teeth, arm, leg and shoulder fossils with other *Homo* species and more primitive ancestors. Previous research had only focused on skull and jaw characteristics.

They found that *H. floresiensis* was far more closely related to *H. habilis* than to *H. erectus* or *H. sapiens*, suggesting it came from an ancient lineage and shared a common ancestor with *H. habilis*. This is reinforced by its more primitive, diminutive body type.

The hobbit's ancestors probably died out across Asia when bigger, more complex human species like *H. erectus* and *H. sapiens* later emerged from Africa, Groves says. *H. floresiensis* was probably only able to cling on in Flores for as long as it did because of its isolation, he says. There's no fossil evidence to indicate that *H. erectus* ever it made it to the island.

So what happened to *H. floresiensis* in the end? The species appears to have died out soon after *H. sapiens* left Africa 60,000 years ago and pushed into Asia. It's possible that a clash between the two species spelled the end of the mysterious Indonesian hobbits.

Journal reference: Journal of Human Evolution, DOI: 10.1016/j.jhevol.2017.02.006

http://bit.ly/2oMosVW

Ancient carvings show comet hit Earth and triggered mini ice age

Ancient symbols carved into stone at an archaeological site in Turkey tell the story of a devastating comet impact that triggered a mini ice age more than 13,000 years ago.

By New Scientist staff and Press Association

Evidence from the carvings, made on a pillar known as the Vulture Stone, suggests that a swarm of comet fragments hit the Earth in around 11000 BC. One image of a headless man is thought to symbolise human disaster and extensive loss of life.

The site is at Gobekli Tepe in southern Turkey, which experts now believe may have been an ancient observatory. Computer software was used to match carvings of animals – interpreted as astronomical symbols – to patterns of stars and pinpoint the event to 10950 BC.

Alistair Coombs/PA Wire

Other evidence for the impact from a Greenland ice core suggests roughly the same time frame. The cataclysm ushered in a cold climate lasting 1,000 years and is likely to have resulted from the break-up of a giant comet in the inner solar system.

'Worst day in history'

"It appears Gobekli Tepe was, among other things, an observatory for monitoring the night sky," says lead researcher Martin Sweatman, from the University of Edinburgh's School of Engineering. "One of its pillars seems to have served as a memorial to this devastating event – probably the worst day in history since the end of the Ice Age."

The carvings appear to have remained important to the people of Gobekli Tepe for millennia, indicating an event that had a very serious and long-lasting impact, say the scientists.

A number of the pillar symbols suggest that long-term changes in the Earth's rotational axis were recorded by the early astronomers using an early form of writing. The discovery also supports the theory that

Earth experiences times when comet strikes are more likely, due to the planet's orbit intersecting with rings of cometary fragments.

Journal reference: Mediterranean Archaeology and Archaeometry, DOI: 10.5281/zenodo.400780

http://bit.ly/2oMxBhv

Cleveland Clinic study finds obesity as top cause of preventable life-years lost

Obesity larger factor than diabetes, tobacco use, hypertension or high cholesterol

A team of researchers from Cleveland Clinic and New York University School of Medicine have found that obesity resulted in as much as 47 percent more life-years lost than tobacco, and tobacco caused similar life-years lost as high blood pressure.

Preliminary work presented by Cleveland Clinic today at the 2017 Society of General Internal Medicine Annual Meeting analyzed the contribution of modifiable behavioral risk factors to causes-of-death in the U.S. population, using 2014 data.

Based on this preliminary work, the team found the greatest number of preventable life-years lost were due to (in order from greatest to least) obesity, diabetes, tobacco use, high blood pressure and high cholesterol. However, researchers also noted that some individuals may have needs that are very different than those of the broader U.S. population. For an obese and alcoholic patient, for example, alcohol use may be more important to address than obesity, even though obesity has a greater impact on the population.

Results highlight the clinical and public health achievement of smoking cessation efforts because 15 years ago, tobacco would have topped the list.

"Modifiable behavioral risk factors pose a substantial mortality burden in the U.S.," said Glen Taksler, Ph.D., internal medicine researcher from Cleveland Clinic and lead author of the study. "These preliminary results continue to highlight the importance of weight loss, diabetes management and healthy eating in the U.S. population."

A key takeaway is that three (diabetes, hypertension and high cholesterol) of the top five causes of death can be treated, so helping patients understand treatment options and approaches can have a powerful impact on life-years. The results also highlight the importance of preventive care in clinical practice and why it should be a priority for physicians.

To estimate the number of life-years lost to each modifiable risk factor, researchers examined the change in mortality for a series of hypothetical U.S. populations that each eliminated a single risk factor. They compared the results with the change in life-years lost for an "optimal" population that eliminated all modifiable risk factors. Recognizing that some less common factors might place substantial burden on small population subgroups, they also estimated life expectancy gained in individuals with each modifiable risk factor.

"The reality is, while we may know the proximate cause of a patient's death, for example, breast cancer or heart attack, we don't always know the contributing factor(s), such as tobacco use, obesity, alcohol and family history. For each major cause of death, we identified a root cause to understand whether there was a way a person could have lived longer."

Dr. Taksler and colleagues are continuing to conduct research in this area, and analyze and refine results.

Research was presented at The Society of General Internal Medicine 2017 Annual Meeting, "Resilience and Grit: Pursuing Organizational Change & Preventing Burnout in GIM" April 19-22, 2017 in Washington, DC at the Washington Hilton Hotel (1919 Connecticut Avenue, NW Washington, DC 20009).

http://bit.ly/2oPtwKb

Earliest Fungus-Like Fossils Date Back 2.4 Billion Years

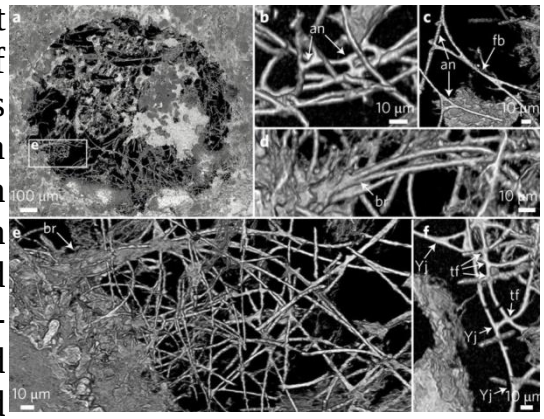
An international group of scientists says it has discovered 2.4 billion-year-old fungus-like fossils

By Jerry Redfern, Seeker | April 24, 2017 04:02pm ET

An international group of scientists says it has discovered 2.4 billion-year-old fungus-like fossils — approximately 2 billion years older than any previous fungal specimen and a billion or more years earlier

than scientists currently think fungi first evolved. If accurate, the finding could reset the spacing of some of the earliest branches on the tree of life.

Birger Rasmussen, a professor at the Western Australian School of Mines, was looking for minerals to date ancient submarine lava collected from bedrock in Northern Cape Province, South Africa, when he found microfilaments in millimeter-sized gas bubbles. "I was startled to find a dense mesh of tangled fossilized microbes," Rasmussen said.



Filamentous fungus-like fossils found in the Ongeluk Formation in South Africa. **Swedish Museum of Natural History**

But gas bubbles in submarine lava can provide a habitat for microorganisms, and knowing that, "we were on the active lookout for fossils in the ancient deep biosphere," said Stefan Bengtson, professor emeritus in paleobiology at the Swedish Museum. He is the lead author of a paper describing the findings, which is published today in Nature Ecology & Evolution. Rasmussen was not looking for the fungus-like structures, "but he had the right mindset to recognize them as fossils," Bengtson said. "It was not accidental."

The South African lava surrounding the fossils was dated at 2.4 billion years old. The structures were found in tiny bubbles and voids within the lava that generally fill with other minerals within 10 million years of forming, Bengtson said, meaning the fossils would be approximately the same age as the rock. "Our organisms had only a limited time to thrive," he said. It is possible, according to Bengtson, that an organism other than fungi formed the structures. "This is why we call the fossils 'fungus-like' rather than 'fungal'," he said. "We have been careful to point out that the filaments we see are very simple."

He described the fossil samples as looking like jumbles of tangled threads that branch and rejoin and said what appear to be bumps along the threads may be spores. There are no known non-fungal equivalents to what was found, Bengtson said.

"[The fossils] are practically indistinguishable in habitus and habitat from the proven fungi in the much younger fossil record," Bengtson said. "We were quite excited that the fossils were so fungus-like."

If the research holds, it would dramatically change "our sense of the timetable of evolutionary history," said Andrew H. Knoll, Fisher Professor of Natural History at Harvard University.

Knoll, however, remains cautious. "Without actually having seen [the research], and giving them the benefit of the doubt, I wouldn't immediately rule out the idea that they are correct in their interpretation," he said. He is skeptical about the timeframe. A fungus is a eukaryote — an organism with a complex cell structure that needs oxygen.

A 2.4 billion-year-old fungus-like eukaryote would have been using oxygen at nearly the same time scientists think oxygen first appeared in notable amounts on the planet. Knoll said he thinks it's likelier the earliest fungi emerged about 1.5 billion years later than the organisms the Swedish group found. "I look forward to seeing [the research] when it comes out and we'll see what happens," he said.

Doug Erwin, curator of Paleozoic invertebrates at the Smithsonian National Museum of Natural History, said he is skeptical.

"[The discovery], if accurate, would be surprising as it would significantly precede fossil evidence and molecular clock analysis for the origin of eukaryotes, much less the origin of fungi," he said.

This is the second major announcement in ancient evolutionary research from Bengtson and the Swedish Museum of Natural History in two months. In March, another group he led announced finding multi-cellular plant fossils in India that they claim pre-dated any other similar specimens by 400 million years.

"Luck," Bengtson said, "favors the prepared mind."

http://bit.ly/2p7s5td

This Very Hungry Caterpillar Eats Plastic Bags

A wiggly, ravenous caterpillar — one that doesn't limit its diet to naturally grown objects — can biodegrade plastic bags, a material infamous for the amount of time it takes to decompose, a new study finds.

By Laura Geggel, Senior Writer | April 24, 2017 05:01pm ET

The 1-inch-long (3 centimeters) wax worm, also known as the honey worm caterpillar (Galleria mellonella), is no stranger to unconventional meals. It's usually found in beehives, munching away on waxy, goo-drenched honeycombs, the researchers said.

Now, through a serendipitous discovery, it's clear that G. mellonella can also decompose polyethylene, a thin but tough plastic that is used across various industries, including in shopping bags and food packaging.

The discovery happened during a beekeeping experience, said the study's senior researcher, Federica Bertocchini, a research scientist at the Spanish National Research Council (CSIC), who also works at the Institute of Biomedicine and Biotechnology of Cantabria, in Santander, Spain. Bertocchini, who is also an amateur beekeeper, happened upon the wax caterpillars when she was cleaning out the panels from one of her beehives. (Beekeeping panels look like wooden picture frames that are filled with honeycomb.)

"I removed the worms, and put them in a plastic bag while I cleaned the panels," Bertocchini said in a statement. "After finishing, I went back to the room where I had left the worms, and I found that they were everywhere. They had escaped from the bag, even though it had been closed." Upon closer inspection, she realized that the caterpillars had made holes in the bag before fleeing. "This project began there and then," Bertocchini said.

When Bertocchini and her colleagues placed the caterpillars on polyethylene plastic bags, holes appeared in the bags within an hour, they found. Perhaps the caterpillars can degrade the plastic because it has chemical bonds that are similar to those found in beeswax, the researchers said. "We have carried out many experiments to test the efficacy of these worms in biodegrading polyethylene," Bertocchini said. "One hundred wax worms are capable of biodegrading 92 milligrams [0.003 ounces] of polyethylene in 12 hours, which really is very fast."

The researchers found that the caterpillars chemically transformed the polyethylene into ethylene glycol. This compound is a colorless and odorless alcohol that has a sweet taste but is poisonous if ingested, according to PubChem, a database at the National Institutes of Health. Ethylene glycol is used as an antifreeze and coolant, PubChem reported.

However, it wasn't clear whether the caterpillar degraded the plastic simply by eating it, the researchers said. So, to find out, they took the caterpillar's whitish cocoon, or chrysalis, and put it against another piece of plastic. Incredibly, the chrysalis also biodegraded the polyethylene, the researchers said.

It's likely that the caterpillars produce an enzyme that can degrade the plastic when they eat it, or when it rubs against them or their chrysalis. The researchers said they hope to detect, isolate and produce it soon on an industrial scale. "In this way, we can begin to successfully eliminate this highly resistant material," Bertocchini said.

Plastic problem

Every year, factories around the world produce about 88 million tons (80 million metric tons) of polyethylene. Although it's used widely — the average person uses about 230 plastic bags annually — the material is slow to degrade. The low-density polyethylene used in plastic bags can take about 100 years to decompose completely, and the most resistant polyethylene products can take up to 400 years to decompose, the researchers said.

Chemical degradation can break down the bags, but this process can take months and uses corrosive liquids, including nitric acid, the researchers said. In contrast, the caterpillar discovery is the first

solution that can biodegrade polyethylene naturally, the researchers said.

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Anti-cancer nutrients in salad leaves increase during postharvest shelf life

Anti-cancer compounds in rocket salad leaves have been found to increase during postharvest shelf life, countering the idea that nutritional content decreases during commercial processing.

Food scientists from the University of Reading have discovered that compounds called isothiocyanates (ITCs) that have properties that fight forms of cancer, including prostate and gastrointestinal cancers, were significantly more abundant one week after processing in species of rocket (Eruca sativa).

In the study, the ITC sulforaphane increased by up to three times following commercial processing and seven days of shelf life.

Dr. Luke Bell, a food chemist from the University of Reading said:

"The discovery is really surprising, going against the assumption that nutrients found in rocket will dissipate over the period of time following harvest.

"Our study has shown that the processing actually has a potentially beneficial effect to consumers, and that rocket lovers can have confidence in the health boost a bag of rocket will give them. The biggest boost in these cancer-fighting compounds came seven days after processing, but begin to tail off after that.

Researchers at the University of Reading also conducted sensory analysis of rocket, to determine if the cancer fighting compounds were more or less prevalent depending on the flavour profile.

Dr. Bell continued:

"We found that there are some distinct varieties of rocket; some that are very hot, and some which are quite mild. The important thing is that the cancer-fighting compounds are prevalent in each variety, meaning that regardless of whether you like rocket mild or hot and peppery, you will still get the same potential health boost."

"With regular consumption of rocket and sulforaphane, consumers could potentially improve their long-term health and reduce the risk of developing other chronic diseases, such as cardiovascular disease."

The study was funded through a BBSRC iCASE award, and partly sponsored by Bakkavor, one of the UK's leading fresh prepared foods supplier; and Elsoms Seeds Ltd. Elsoms providing the seeds for experiments and Bakkavor provided use of their facilities for growing field trials and processing the rocket leaves, and both companies provided a financial contribution.

Dr Lorraine Shaw, Agronomic Development Technologist from Bakkavor said:

"***As a leading supplier of prepared salads we are keen to support research projects such as this. It helps us to further understand the role key ingredients can play in the healthy eating habits of consumers".***

Robin Wood, Managing Director of Elsoms Seeds, added "This has been a valuable study as part of our ongoing research and development focusing on the properties and flavours of herbs. We want to understand how we can improve the taste sensation of rocket for the benefit of the consumer so we were delighted to support the project which has given us valuable information for our rocket breeding programme."

More information: Luke Bell et al. Changes in rocket salad phytochemicals within the commercial supply chain: Glucosinolates, isothiocyanates, amino acids and bacterial load increase significantly after processing, Food Chemistry (2017). DOI: 10.1016/j.foodchem.2016.11.154

http://bit.ly/2plV6ja

Patients with drug-resistant malaria cured by plant therapy developed at WPI

Tablets made from dried leaves of the Artemisia annua plant cured 18 critically ill patients in a Congo clinic. The results suggest a new and inexpensive treatment option for the mosquito-borne disease that affects 212 million people worldwide

Worcester, Mass. - When the standard malaria medications failed to help 18 critically ill patients, the attending physician in a Congo clinic acted

under the "compassionate use" doctrine and prescribed a not-yet-approved malaria therapy made only from the dried leaves of the Artemisia annua plant. In just five days, all 18 people fully recovered. This small but stunningly successful trial offers hope to address the growing problem of drug-resistant malaria.

Details of the cases are documented in the paper "Artemisia annua dried leaf tablets treated malaria resistant to ACT and i.v. artesunate: case reports" by an international team lead by Pamela Weathers, PhD, professor of biology and biotechnology at Worcester Polytechnic Institute (WPI), who has pioneered the use of dried leaves of Artemisia annua (DLA) as a malaria therapy.

"To our knowledge, this is the first report of dried-leaf Artemisia annua controlling ACT-resistant malaria in humans," the authors of the Phytomedicine paper note, adding that more comprehensive clinical trials on patients with drug-resistant malaria are warranted. "Successful treatment of all 18 ACT-resistant cases suggests that DLA should be rapidly incorporated into the antimalarial regimen for Africa," they added, "and possibly wherever else ACT resistance has emerged."

Watch a video about research at WPI related to this study.

The report documents the experiences of 18 patients in the North Kivu province of the Democratic Republic of Congo who showed symptoms of malaria and were originally treated with the recommended medication: artemisinin-based combination therapy (ACT), which blends artemisinin, a chemical extract from Artemisia annua, with one or more other drugs that attack the malaria parasite in different ways.

The 18 patients, ranging in age from 14 months to 60 years, did not respond to the standard ACT treatment, and all lapsed into severe malaria, defined by symptoms that can include loss of consciousness, respiratory distress, convulsions, and pulmonary edema. One patient, a five-year-old child, became comatose. All were then treated with intravenously administered artesunate, the frontline medication for severe malaria, but again they showed no improvement.

As a last resort, doctors turned to dried-leaf Artemisia (DLA), a therapy developed and extensively studied by Weathers and her team at WPI. After five days of treatment with tablets made from only the dried and powered leaves of Artemisia (which has been prepared and analyzed using methods developed by Weathers and postdoctoral fellow Melissa Towler), all 18 patients fully recovered. Laboratory tests showed they had no parasites remaining in their blood. (Weathers noted more than 100 other drug-resistant patients also have been successfully treated with DLA tablets.)

"These 18 patients were dying," Weathers said. "So to see 100 percent recover, even the child who had lapsed into a coma, was just amazing. It's a small study, but the results are powerful."

According to the World Health Organization (WHO), more than 212 million people contracted malaria in 2015 and some 429,000 died, with young children and pregnant women being particularly vulnerable. Caused by a mosquito-borne parasite, the illness is reported in nearly 100 countries and threatens nearly half of the world's population.

ACT, the current recommended therapy, is expensive to produce and is in short supply in areas hit hardest by the disease. In addition, while the combination therapy is designed to be less prone to the drug resistance that has rendered previous antimalarial agents ineffective, increasingly the malaria parasite is showing signs of resistance to ACT, particularly in Southeast Asia.

Weathers began her research on artemisinin and Artemesia annua (also known as sweet wormwood) more than 25 years ago. In recent years, she has turned her attention to the use of DLA as an alternative to conventional antimalarial drugs. Noting that Artemisia annua, which is classified as a generally regarded as safe (GRAS) herb, has been consumed by humans and used as an herbal therapy for thousands of years, often in the form of a tea, she became intrigued by

the potential for using the dried plant, rather than just a chemical extract, as a malaria treatment.

A study she published in Photochemistry Reviews in 2011 was the first to demonstrate that dried leaves of the Artemisia annua plant delivers 40 times more artemisinin to the blood than does the drug based on the chemical extract of the plant.

In a paper published in PLOS ONE the following year, Weathers and her team showed that not only does DLA have antimalarial properties, it is more effective in knocking out the parasite and reduced the level of parasite infection more completely in mice.

In a 2015 study in the Proceedings of the National Academy of Sciences, the WPI researchers, with colleagues at the University of Massachusetts Amherst, showed that dried Artemisia leaves cured rodents infected with malaria strains that were known to be resistant to artemisinin. And, in an experiment that accelerated the evolution of the malaria parasite by passing it through up to 49 generations of mice, the parasite showed no signs of resistance to DLA.

Weathers says the superior performance of DLA in comparison to ACT, as well as its ability to kill drug-resistant parasites and avoid the resistance trap, itself, is likely due to the synergistic effects of a complex array of phytochemicals contained in the plant's leaves, several of which are also known to have antimalarial properties and others of which may act both to enhance the absorption of artemisinin into the bloodstream and bolster its effectiveness against malaria. In effect, the dried leaves constitute a robust natural combination therapy, one whose benefits far surpass those of ACT and other combination drugs.

"We have done a lot of work to understand the biochemistry of these compounds, which include a number of flavonoids and terpenes, so we can better understand the role they play in the pharmacological activity of the dried leaves," Weathers said. "The more we learn, the more excited we become about the potential for DLA to be the medication of choice for combatting malaria worldwide. Artemisia annua is known to be efficacious against a range of other diseases, including other tropical maladies and certain cancers, so in our lab we are already at work investigating the effectiveness of DLA with other diseases."

Another advantage of DLA over conventional malaria treatments is its low cost and the relative simplicity of its manufacture, Weathers said. While the processes for manufacturing ACT is costlier and requires a higher degree of expertise, producing DLA tablets can be accomplished with simpler equipment and a modest amount of training. Growing Artemisia annua and producing and testing the tablets, Weathers noted, are ideal local business that can provide jobs in impoverished areas and greatly expand access to antimalarial therapy.

In fact, she has already established a supply chain in Africa that includes growing and harvesting high-producing cultivars in East Africa, along with GMP (Good Manufacturing Practice) processing operations in Uganda where the leaves are dried, pulverized, and homogenized, where the powder is compacted into tablets, and where the tablets are tested to verify their dosage. This supply chain helped produce the tablets used to treat the 18 patients in the Democratic Republic of Congo.

"This simple technology can be owned, operated, and distributed by Africans for Africans," Weathers said.

http://bit.ly/2qkrd2U

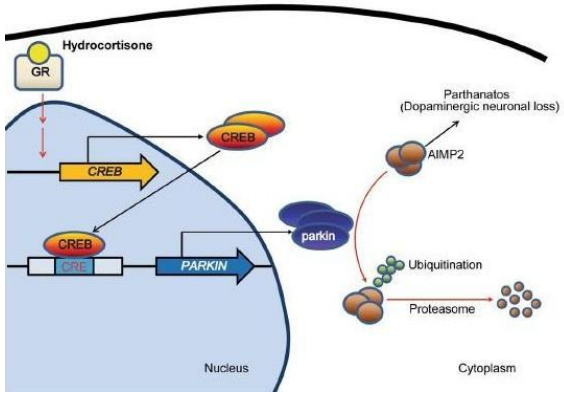
Parkinson's disease will be curable with cortisol

DGIST's research team has found a candidate substance which can prevent and cure Parkinson's disease.

By using this substance, the team also has identified the mechanism of dopaminergic neuronal death inhibition.

Parkinson's disease is a representative neurological degenerative brain disease caused by the death of dopaminergic neurons in the middle cerebral blood. It is a disease with high incidence in the population

over the age of 60 and the symptoms are body tremor and stiffness, slow motion, posture instability, etc.



It is known that mutation or low expression of parkin protein, a part of the system which hydrolyzes intracellular proteins, accelerates the accumulation of toxic proteins that must be removed in cells and induces dopaminergic neuronal cell death and Parkinson's disease, a degenerative brain disease.

Hydrocortisone binds to glucocorticoid receptor which in turn leads to expression of CREB. CREB increases parkin expression via binding to CREB binding motifs of parkin promoter region. Hydrocortisone-stimulated parkin expression results in the downregulation of the toxic parkin substrate AIMP2, which is beneficial for dopaminergic neuronal survival. **DGIST**

Currently, Parkinson's disease is classified as a rare incurable disease, one of the Korean government's four major target serious illnesses. However, there are no drugs that can prevent the death of dopaminergic neurons.

The senior researcher Yoon-Il Lee's research team and Professor Yunjong Lee's research team have continuously conducted studies on the development of candidate substances to cure Parkinson's disease and their mechanisms. The researchers performed a high-throughput screening method to identify drug candidates that promote dopaminergic neuronal cell activation by inducing the expression of the parkin protein, the cell protection gene which can inhibit the death of dopaminergic neurons.

As a result, it has been identified that cortisol* , known as a stress hormone, induces the expression of the parkin protein and prevents dopaminergic neuronal death by eliminating the accumulation of cell death factors through ubiquitin proteasome system.

In addition, the team has demonstrated the mechanism by which cortisol induces the expression of the parkin protein and CREB transcriptional regulator through the hormone receptor regulates the expression of the parkin protein through the cell and animal model experiments. The study also has assured the possibility that cortisol can be used as a therapeutic agent for degenerative Parkinson's disease.

The senior researcher Yoon-Il Lee stated "The significance of this study is that it has identified that the expression of parkin protein induced by a moderate level of stress hormone cortisol could be an important factor in maintaining the viability of dopaminergic neurons. We will continue to conduct follow-up studies such as clinical studies so that the Parkinson's disease will be curable in the future."

http://bit.ly/2oU8sRT

Hungry stomach hormone promotes growth of new brain cells

Could fasting boost your brainpower?

By Clare Wilson

A stomach hormone that stimulates appetite seems to promote the growth of new brain cells and protect them from the effects of ageing – and may explain why some people say that fasting makes them feel mentally sharper.

When ghrelin was first discovered, it became known as the hunger hormone. It is made by the stomach when it gets empty, and whenever we go a few hours without food its levels rise in our blood.

But there is also evidence that ghrelin can enhance cognition. Animals that have reduced-calorie diets have better mental abilities, and ghrelin might be part of the reason why. Injecting the hormone into mice improves their performance in learning and memory tests, and seems to boost the number of neuron connections in their brains.

Now Jeffrey Davies at Swansea University, UK, and his team have found further evidence that ghrelin can stimulate brain cells to divide and multiply, a process called neurogenesis. When they added the

hormone to mouse brain cells grown in a dish, it switched on a gene known to trigger neurogenesis, called fibroblast growth factor.

New memories

If the same effect happens in animals, this could be how ghrelin exerts its effects on memory, says Davies, whose work was presented at the British Neuroscience Association conference this month. Young brain cells are thought to enhance the ability to form new memories. This is because they are more excitable, so are more likely to be activated by new environments. “These neurons will fire more easily than old neurons, and they set in play a new memory,” says Davies.

The work may also have implications for treating neurodegenerative conditions, such as Parkinson’s disease, which is caused by a loss of a type of brain cell. Previous research, including some by Davies’s team, has found that ghrelin can help protect animals from developing a form of Parkinson’s disease.

In further experiments, Davies’s team found that ghrelin protects brain cells in a dish from dying when they are encouraged to mimic Parkinson’s disease. And Davies’s colleague Amanda Hornsby found that, in a study of 28 volunteers, people with Parkinson’s dementia – cognitive impairment caused by Parkinson’s disease – have lower levels of ghrelin in their blood than people who don’t have the condition. This suggests that ghrelin, or other chemicals that act the same way, could be used as a treatment for Parkinson’s dementia, says Hornsby.

Intermittent fasting

In people, going on a permanent diet of about 25 per cent fewer calories than the daily recommended amount has several benefits to health, such as better control of blood sugar levels. Some who try it have said it also improves their cognitive abilities, although this is controversial – some studies have suggested it impairs people’s mental abilities.

In an effort to harness some of the health benefits of a calorie-restricted diet, some people are turning to intermittent fasting. It’s likely, for example, that the 5-2 diet, where people eat normally for five days but stick to about 500 calories a day for the other two, raises ghrelin levels.

But Nicolas Kunath of the Technical University of Munich, Germany, points out that new brain cells take a few days to weeks to start working, so people shouldn’t expect fasting to produce immediate effects on their brainpower in this way.

http://bit.ly/2oKVU48

A more than 100% quantum step toward producing hydrogen fuel

Key breakthrough in basic science essential for progress toward efficient production of gaseous hydrogen fuel using solar energy

Efforts to reduce our dependence on fossil fuels are advancing on various significant fronts. Such initiatives include research focused on more efficient production of gaseous hydrogen fuel by using solar energy to break water down into its components of hydrogen and oxygen. Recently, in an article published in the journal Nature Energy, lead author Yong Yan, an assistant professor in the Department of Chemistry and Environmental Science, reported a key breakthrough in the basic science essential for progress toward this goal.

The article, "Multiple exciton generation for photoelectrochemical hydrogen evolution reactions with quantum yields exceeding 100%," reports on the investigative work that Yan carried out along with colleagues affiliated with the National Renewable Energy Laboratory, the Colorado School of Mines and San Diego State University. Essentially, they created what is known as a quantum dot photoelectrochemical cell that catalytically achieved quantum efficiency for hydrogen gas production exceeding 100% -- in the case of their experiments an efficiency approaching 114%.

Quantum dots are extremely small semiconductor particles only a few nanometers in size. (A nanometer is one-billionth of a meter.) In their device, lead sulfide quantum dots replace semiconductor materials such as silicon and copper indium gallium arsenide. The advantage is

that such a photoelectrochemical device can, potentially, convert a greater portion of the solar spectrum into useful energy.

The device described is able to absorb one visible solar photon and produce two, or even more, electrons through a process known as multiple exciton generation, or MEG, which are further utilized to reduce water to generate hydrogen gas. Although many scientists worldwide are engaged in efforts to achieve quantum efficiency as close as possible to 100% for solar hydrogen production, Yan's achievement in directly exceeding this threshold is a significant fundamental breakthrough. It clearly proves that the photoelectrochemical cell design he describes is much more efficient than a quantum dot solar cell with respect to quantum yield.

Yan, who joined the NJIT faculty in 2016, emphasizes that this advance is at the level of basic solar science, and that the breakthrough with respect to quantum yield does not equate to a substantial increase in the ultimate solar-to-hydrogen conversion efficiency. Nonetheless, this dramatic increase in quantum yield realized with a uniquely innovative lead sulfide quantum dot photoelectrochemical device is an important development in several ways, and as such is a product of Yan's long-standing interest in renewable sources of energy, especially in novel applications of solar energy.

For Yan, the research reported in Nature Energy culminated at NJIT after his previous work as a postdoc at Princeton University and at the U.S. Department of Energy's National Renewable Energy Laboratory in Colorado. The success of this leading-edge effort was made possible with funding provided, in part, by NJIT and the Department of Energy.

Yan says, "These results do present the possibility of generating more energy more efficiently with such a solar-capture device in the future. This could also lead to a fundamental change in the entire process of producing hydrogen fuel. We can now obtain hydrogen fuel from water by using electricity supplied by conventional power plants that consume fossil fuels. But by building on the basic step of achieving such high quantum efficiency for solar hydrogen generation, we could make the process of producing a 'green' fuel much greener as well."

http://bit.ly/2qhrjv7

Popular belief that saturated fats clog up arteries 'plain wrong' say experts

Best form of prevention and treatment are 'real' food and a brisk 22 minute daily walk

The widely held belief among doctors and the public that saturated fats clog up the arteries, and so cause coronary heart disease, is just "plain wrong," contend experts in an editorial published online in the British Journal of Sports Medicine.

It's time to shift the focus away from lowering blood fats and cutting out dietary saturated fat, to instead emphasising the importance of eating "real food," taking a brisk daily walk, and minimising stress to stave off heart disease, they insist.

Coronary artery heart disease is a chronic inflammatory condition which responds to a Mediterranean style diet rich in the anti-inflammatory compounds found in nuts, extra virgin olive oil, vegetables and oily fish, they emphasise.

In support of their argument Cardiologists Dr Aseem Malhotra, of Lister Hospital, Stevenage, Professor Rita Redberg of UCSF School of Medicine, San Francisco (editor of JAMA Internal medicine) and Pascal Meier of University Hospital Geneva and University College London (editor of BMJ Open Heart) cite evidence reviews showing no association between consumption of saturated fat and heightened risk of cardiovascular disease, diabetes, and death.

And the limitations of the current 'plumbing theory' are writ large in a series of clinical trials showing that inserting a stent (stainless steel mesh) to widen narrowed arteries fails to reduce the risk of heart attack or death, they say.

"Decades of emphasis on the primacy of lowering plasma cholesterol, as if this was an end in itself and driving a market of 'proven to lower

cholesterol' and 'low fat' foods and medications, has been misguided," they contend. Selective reporting of the data may account for these misconceptions, they suggest.

A high total cholesterol to high density lipoprotein (HDL) ratio is the best predictor of cardiovascular disease risk, rather than low density lipoprotein (LDL). And this ratio can be rapidly reduced with dietary changes such as replacing refined carbohydrates with healthy high fat foods (such as nuts and olive oil), they say.

A key aspect of coronary heart disease prevention is exercise, and a little goes a long way, they say. Just 30 minutes of moderate activity a day three or more times a week works wonders for reducing biological risk factors for sedentary adults, they point out. And the impact of chronic stress should not be overlooked because it puts the body's inflammatory response on permanent high alert, they say.

All in all, a healthy diet, regular exercise, and stress reduction will not only boost quality of life but will curb the risk of death from cardiovascular disease and all causes, they insist.

"It is time to shift the public health message in the prevention and treatment of coronary artery disease away from measuring serum lipids and reducing dietary saturated fat," they write.

"Coronary artery disease is a chronic inflammatory disease and it can be reduced effectively by walking 22 minutes a day and eating real food." But, they point out: "There is no business model or market to help spread this simple yet powerful intervention."

http://bit.ly/2qrPBPr

Homo naledi is only 250,000 years old – here's why that matters

Implications of the announcement that* H. naledi *fossils are between 300,000 and 200,000 years old

By Colin Barras

In 2013, Lee Berger at the University of the Witwatersrand in Johannesburg and his colleagues made an extraordinary discovery – deep inside a South African cave system they found thousands of bones belonging to a brand new species of early human - and now we finally may know when this species lived and how it fits into our evolutionary tree.

By 2015 it was becoming clear that the new species, which was named *Homo naledi*, was unlike anything researchers had discovered before. Although parts of its skeleton looked identical to our modern human anatomy, it had some features that were strikingly primitive – including a skull that was only slightly larger than that of a chimpanzee.

Stefan Heunis/AFP/Getty Images

But Berger and his colleagues had trouble establishing how old the *H. naledi* fossils were. Without that piece of information, most other researchers agreed that the true significance of *H. naledi* for understanding human evolution was unclear. Guesses have varied from as old as 2 million years to as young as 100,000 years.

Today, news broke that Berger's team has finally found a way to date the fossils. In an interview published by *National Geographic* magazine, Berger revealed that the *H. naledi* fossils are between 300,000 and 200,000 years old.

"This is astonishingly young for a species that still displays primitive characteristics found in fossils about 2 million years old, such as the small brain size, curved fingers, and form of the shoulder, trunk and hip joint," says Chris Stringer at the Natural History Museum in London. Here, we address some of the implications of the announcement, as we wait for the full publication of the results.

Why has it taken so long to establish the age of the fossils?

It can be surprisingly difficult to work out how old fossil bones are. Many of the techniques researchers can use require the isotopic

analysis of bone samples. Berger and his colleagues are reluctant to use these techniques, because they involve destroying small samples of precious fossil material.

Another option is to date the rock or sediment that blankets the layer in which the fossils are found. Ancient lava flows, in particular, contain chemical signatures that are perfect for isotopic dating. But the *H. naledi* remains were found in a cave in which there were no easily dated sedimentary layers covering the fossils.

Researchers can also work out the rough age of the fossils by looking at the fossil remains of other species found alongside them, if the age of those other species has already been established. The cave in which the *H. naledi* fossils were found contains virtually no bones from other species, though, making this approach a nonstarter.

So how did Berger and his colleagues work out the age of the fossils?

We don't know yet. The scientific papers in which this information will be revealed haven't been published. The *National Geographic* interview mentions that Berger and his colleagues have found a second cave chamber containing more *H. naledi* remains – perhaps these additional fossils were preserved in a context that made dating less challenging.

If the fossils are 300,000 to 200,000 years old what does that mean?

Our earliest hominin ancestors lived at least seven million years ago. The first species to look a little like modern humans appeared between about two and three million years ago. But our own species – *Homo sapiens* – evolved about 200,000 years ago. So, if *H. naledi* lived 300,000 to 200,000 years ago that's a remarkable discovery.

It means that a species of human with some surprisingly primitive features – including a tiny skull and brain – survived into the relatively recent past. Conceivably, *H. naledi* might even have met early members of our species, *H. sapiens*. One could even speculate we had something to do with it going extinct.

Does the age help us to work out where *H. naledi* fits in the human evolutionary tree?

It probably depends on whom you ask. Based purely on its strange anatomy, *H. naledi* seems to belong somewhere near the very base of the "true human" family tree – an idea suggested in some studies of the fossils.

But we know that the first early humans appeared more than two million years ago. If *H. naledi* is just 300,000 years old, some researchers might argue that it can't belong to the base of our family tree. It's too young. Perhaps it even had a modern-looking ancestor and later evolved primitive-looking features.

But it is, in fact, still perfectly possible that *H. naledi* really does belong somewhere near the base of our human evolutionary tree.

The species might have evolved more than two million years ago, as one of the earliest "true" humans, and then survived, unchanged, for hundreds of thousands of years. "It could lie close to the origin of the genus *Homo*, suggesting that this is a relic species, retaining many primitive traits from a much earlier time," says Stringer.

Berger has previously talked about this possibility. He says *H. naledi* might be like a human version of the coelacanth – a primitive fish with ancestors that first appeared 400 million years ago but that is still found in oceans today.

Is there any precedent for that idea in the human fossil record?

Yes – potentially. About a decade ago researchers working on the opposite side of the world, in Indonesia, made another astonishing discovery: they found remains of another ancient human species with a tiny chimp-sized head that also lived just a few hundred thousand years ago. It is named *Homo floresiensis* – although it is better known by its nickname: the "hobbit".

Researchers have been arguing about *H. floresiensis*'s place in the human family tree for years. Last week, one paper revived the idea that *H. floresiensis* can trace its roots back to a very early species of

human called *H. habilis* that we know lived in Africa more than two million years ago.

The idea is that a population of *H. habilis* left Africa about two million years ago and gradually moved across Asia, ultimately reaching Indonesia. If this idea is correct, *H. floresiensis* falls on one of the lowest branches in the "true" human family tree despite its young age, because it evolved directly from the primitive *H. habilis*.

In other words, species of evolutionarily primitive humans might, in some circumstances, be able to survive for hundreds of thousands of years.

"There are obvious parallels with the late survival of *H. floresiensis* in Indonesia, but in that case island isolation probably accounts for its longevity," says Stringer. "How did a comparably strange and small-brained species linger on in southern Africa, seemingly alongside more 'advanced' humans?"

What happened to *H. naledi* in the end?

There are no answers to this question yet. But if the fossils really are just 300,000 to 200,000 years old there is at least one possible scenario. Our species, *H. sapiens*, evolved in Africa about 200,000 years ago. If those early *H. sapiens* reached southern Africa shortly afterwards, they might have contributed to the extinction of *H. naledi*.

Again there is precedent for this. The fossil record elsewhere in the world shows that *H. sapiens* left Africa and gradually spread across Eurasia. As it did so, *H. sapiens* arrived in areas already populated by ancient humans – species like the Neanderthals. Within a few thousand years of *H. sapiens* arriving in these new areas, the indigenous species of ancient humans disappeared, apparently outcompeted by *H. sapiens*.

Even the hobbit, *H. floresiensis*, seems to have suffered this fate. The most recent information suggests it went extinct 50,000 years ago – about the same time that *H. sapiens* arrived in this part of Indonesia. *H. naledi* might have the dubious honour of being the earliest ancient human species to have been driven to extinction by the spread of our species. But this is still speculation at the moment.

http://bit.ly/2ptN6wx

Researchers map the evolution of dog breeds

Researchers have used gene sequences from 161 modern breeds to assemble an evolutionary tree of dogs

When people migrate, Canis familiaris travels with them. Piecing together the details of those migrations has proved difficult because the clues are scattered across the genomes of hundreds of dog breeds. However, in a study published April 25 in Cell Reports, researchers have used gene sequences from 161 modern breeds to assemble an evolutionary tree of dogs.

This photograph shows a toy xoloitzcuintlel, a dog breed that likely descended from dogs that crossed the Bering Land Bridge with Native Americans Ancestors. **Penny Inman**

The map of dog breeds, which is the largest to date, unearths new evidence that dogs traveled with humans across the Bering land bridge, and will likely help researchers identify disease-causing genes in both dogs and humans.

The study highlights how the oldest dog breeds evolved or were bred to fill certain roles. "First, there was selection for a type, like herders or pointers, and then there was admixture to get certain physical traits," says study co-author and dog geneticist Heidi Parker of the National Institutes of Health (NIH). "I think that understanding that types go back a lot longer than breeds or just physical appearances do is something to really think about."

Most popular breeds in America are of European descent, but in the study, researchers found evidence that some breeds from Central and South America—such as the Peruvian Hairless Dog and the Xoloitzcuintle—are likely descended from the "New World Dog," an

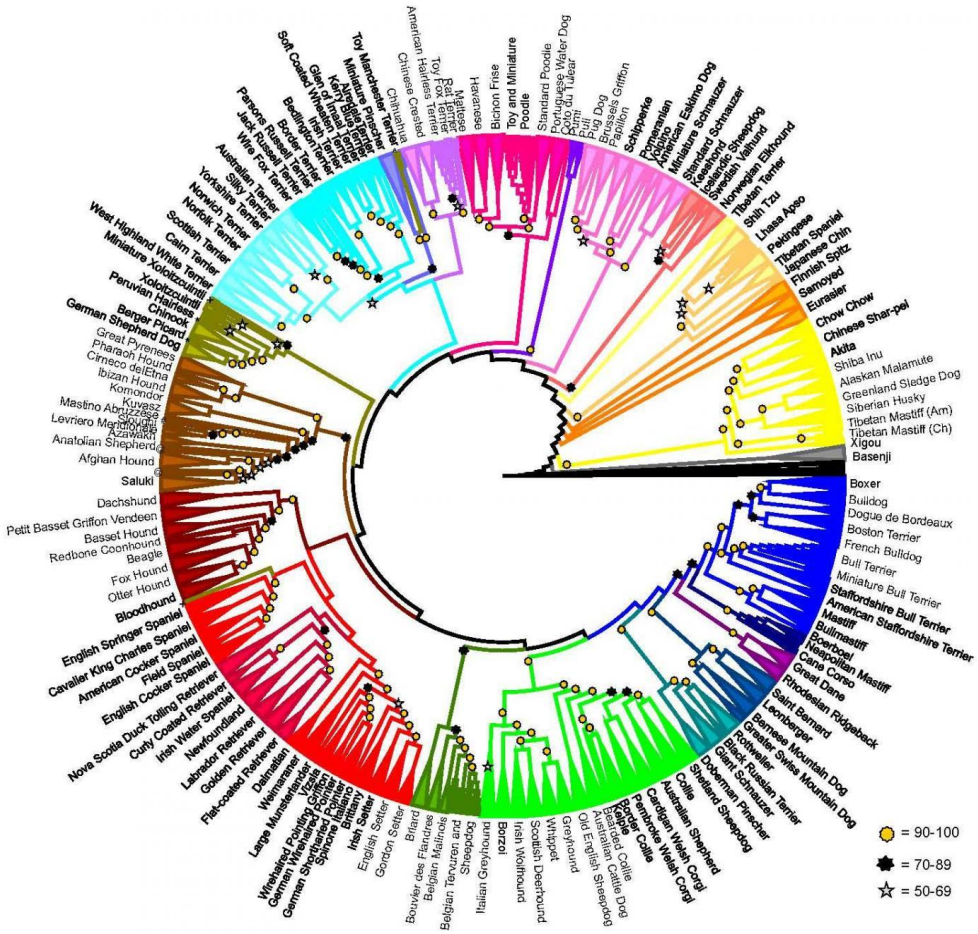
ancient canine sub-species that migrated across the Bering Strait with the ancestors of Native Americans. Scientists have previously reported archaeological evidence that the New World Dog existed, but this study marks the first living evidence of them in modern breeds.

"What we noticed is that there are groups of American dogs that separated somewhat from the European breeds," says study co-author Heidi Parker of the NIH. "We've been looking for some kind of signature of the New World Dog, and these dogs have New World Dogs hidden in their genome." It's unclear precisely which genes in modern hairless dogs are from Europe and which are from their New World ancestors, but the researchers hope to explore that in future studies.

Other results were more expected. For instance, many breeds of "gun dogs," such as Golden Retrievers and Irish Setters, can trace their origins to Victorian England, when new technologies, such as guns, opened up new roles on hunting expeditions. Those dogs clustered closely together on the phylogenetic tree, as did the spaniel breeds. Breeds from the Middle East, such as the Saluki, and from Asia, such as Chow Chows and Akitas, seem to have diverged well before the "Victorian Explosion" in Europe and the United States.

Herding breeds, though largely European in origin, proved to be surprisingly diverse. "When we were looking at herding breeds, we saw much more diversity, where there was a particular group of herding breeds that seemed to come out of the United Kingdom, a particular group that came out of northern Europe, and a different group that came out of southern Europe," says Parker, "which shows herding is not a recent thing. People were using dogs as workers thousands of years ago, not just hundreds of years ago."

Different herding dogs use very different strategies to bring their flocks to heel, so in some ways, the phylogenetic data confirmed what many dog experts had previously suspected, the researchers noted. "What that also tells us is that herding dogs were developed not from a singular founder but in several different places and probably different times," says the study's senior co-author and dog geneticist Elaine Ostrander, also of the NIH.



This evolutionary tree shows the relationships between dog breeds. **NIH Dog Genome Project**

Ostrander and her colleagues have spent years sequencing dog genomes but can also frequently be found out in the field at dog shows, recruiting dog owners to participate in the study. "If we see a breed that we haven't had a good sample of to sequence, we definitely make a beeline for that owner," says Ostrander. "And say, 'Gosh, we don't have the sequence of the Otterhound yet, and your dog is a beautiful

Otterhound. Wouldn't you like it to represent your breed in the dog genome sequence database?' And of course, people are always very flattered to say, "Yes. I want my dog to represent Otterhound-ness." All of the dog sequences in the study are from dogs whose owners volunteered, Ostrander says. Over half the dog breeds in the world today still have not been sequenced and the researchers intend to keep collecting dog genomes to fill in the gaps.

Understanding dogs' genetic backstory also has practical applications. Our canine compatriots fall victim to many of the same diseases that humans do—including epilepsy, diabetes, kidney disease, and cancer—but disease prevalence varies widely and predictably between breeds, while it is more difficult to compartmentalize at-risk human populations. "Using all this data, you can follow the migration of disease alleles and predict where they are likely to pop up next, and that's just so empowering for our field because a dog is such a great model for many human diseases," says Ostrander. "Every time there's a disease gene found in dogs it turns out to be important in people, too."

Cell Reports, Parker et al.: "Genomic Analyses Reveal the Influence of Geographic Origin, Migration, and Hybridization on Modern Dog Breed Development" http://www.cell.com/cell-reports/fulltext/S2211-1247(17)30456-4 , DOI: 10.1016/j.celrep.2017.03.079

http://bit.ly/2oLCKuP

Could Parkinson's disease start in the gut?

Parkinson's disease may start in the gut and spread to the brain via the vagus nerve

MINNEAPOLIS - Parkinson's disease may start in the gut and spread to the brain via the vagus nerve, according to a study published in the April 26, 2017, online issue of Neurology®, the medical journal of the American Academy of Neurology. The vagus nerve extends from the brainstem to the abdomen and controls unconscious body processes like heart rate and food digestion.

The preliminary study examined people who had resection surgery, removing the main trunk or branches of the vagus nerve. The surgery, called vagotomy, is used for people with ulcers. Researchers used national registers in Sweden to compare 9,430 people who had a vagotomy over a 40-year period to 377,200 people from the general population. During that time, 101 people who had a vagotomy developed Parkinson's disease, or 1.07 percent, compared to 4,829 people in the control group, or 1.28 percent. This difference was not significant.

But when researchers analyzed the results for the two different types of vagotomy surgery, they found that people who had a truncal vagotomy at least five years earlier were less likely to develop Parkinson's disease than those who had not had the surgery and had been followed for at least five years. In a truncal vagotomy, the nerve trunk is fully resected. In a selective vagotomy, only some branches of the nerve are resected.

A total of 19 people who had truncal vagotomy at least five years earlier developed the disease, or 0.78 percent, compared to 3,932 people who had no surgery and had been followed for at least five years, at 1.15 percent. By contrast, 60 people who had selective vagotomy five years earlier developed Parkinson's disease, or 1.08 percent.

After adjusting for factors such as chronic obstructive pulmonary disease, diabetes, arthritis and other conditions, researchers found that people who had a truncal vagotomy at least five years before were 40 percent less likely to develop Parkinson's disease than those who had not had the surgery and had been followed for at least five years.

"These results provide preliminary evidence that Parkinson's disease may start in the gut," said study author Bojing Liu, MSc, of the Karolinska Instituet in Stockholm, Sweden. "Other evidence for this hypothesis is that people with Parkinson's disease often have gastrointestinal problems such as constipation, that can start decades before they develop the disease. In addition, other studies have shown that people who will later develop Parkinson's disease have a protein believed to play a key role in Parkinson's disease in their gut."

The theory is that these proteins can fold in the wrong way and spread that mistake from cell to cell.

"Much more research is needed to test this theory and to help us understand the role this may play in the development of Parkinson's," Liu said. Additionally, since Parkinson's is a syndrome, there may be multiple causes and pathways.

Even though the study was large, Liu said one limitation was small numbers in certain subgroups. Also, the researchers could not control for all potential factors that could affect the risk of Parkinson's disease, such as smoking, coffee drinking or genetics.

The study was supported by the Swedish Research Council for Health, Working Life and Welfare, the Parkinson Research Foundation in Sweden, and the U.S. National Institutes of Health.

http://bit.ly/2qezRPB

Lawsuit: Mylan's epic EpiPen price hike wasn't about greed—it's worse

With higher prices, Mylan allegedly dangled deep discounts—if buyers excluded rival.

Beth Mole - 4/26/2017, 6:58 AM

When Mylan dramatically increased the price of its life-saving EpiPen devices, it drew sharp rebuke all around for what seemed like a purely greedy—and heartless—move. But according to a lawsuit filed by French drug maker Sanofi, the move wasn't just out of simple greed. Instead, it was part of an underhanded scheme to "squash" competition from Sanofi's rival device, the Auvi-Q.

With the lofty prices and near-monopoly over the market, Mylan could dangle deep discounts to drug suppliers—with the condition that they turn their backs on Sanofi's Auvi-Q—the lawsuit alleges. Suppliers wouldn't dare ditch EpiPens, the most popular auto-injector. And with the high prices, the rebates wouldn't put a dent in Mylan's hefty profits, Sanofi speculates.

Coupled with a smear campaign and other underhanded practices, Mylan effectively pushed Sanofi out of the US epinephrine auto-injector market, Sanofi alleges. The lawsuit, filed Monday in a federal court in New Jersey, seeks damages under US Antitrust laws.

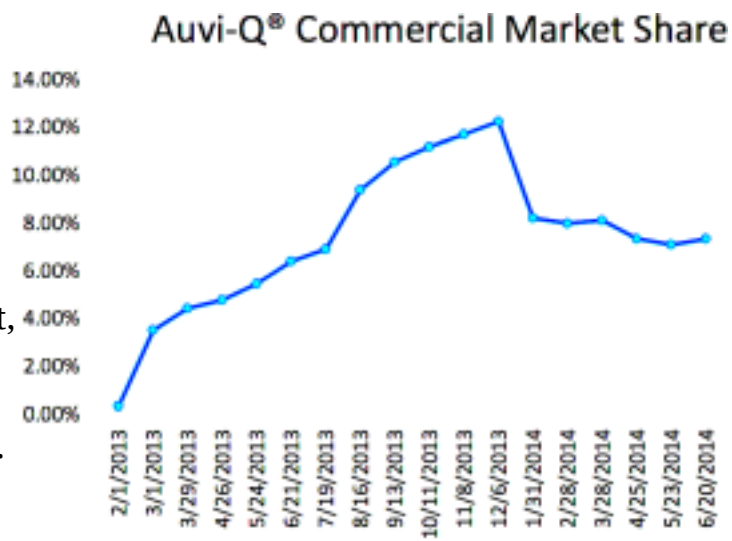
In 2013, Sanofi began selling Auvi-Q, which works to quell life-threatening allergic reactions, just as EpiPen does. Sanofi priced Auvi-Q on equal footing with EpiPen. And, initially, Sanofi claims it showed promise of gaining market share and providing real competition to EpiPen, which at the time had more than 99 percent of the market, according to Mylan. But that all changed when Mylan began using dirty tactics, Sanofi alleges.

In short, Sanofi claims that "Mylan engaged in illegal conduct to squelch this nascent competition, harming both Sanofi and U.S. consumers."

According to the lawsuit:

In particular, Mylan offered new and unprecedented rebates to commercial insurance companies, pharmaceutical benefit managers, and state-based Medicaid agencies (collectively "third-party payors") conditioned exclusively on Auvi-Q® not being an [epinephrine auto-injector] drug device that those payors would reimburse for use by U.S. consumers.

Sanofi alleges that Mylan's rebates jumped from less than 10 percent to 30 percent or higher. And as a consequence to third-parties taking the bait, Sanofi was blocked from nearly 50 percent of the market. When the rebates fully kicked in by December 2013, Sanofi's market share sharply dropped from nearly 13 percent to 8 percent and then to 7 percent.



Meanwhile, Mylan used smear tactics, producing a study that seemed to question the Food and Drug Administration's decision to deem Auvi-Q "bioequivalent" to EpiPen, Sanofi alleges.

Sanofi notes that the tactics are in line with other underhanded efforts by Mylan, including misclassifying EpiPen to regulators, which cheated federal and state governments out of millions, and making deals to provide schools with EpiPen if they agreed not to use rival devices. Mylan later removed the condition from deals with schools when it was publicized, which Sanofi alleges is tantamount to admission of guilt.

To demonstrate the overall effects of Mylan's efforts, Sanofi points to data comparing Auvi-Q's market share in the US with that in Canada. By 2014, Auvi-Q still lingered around 7 percent in the US, while it neared 25 percent across the northern border.

In 2015, Sanofi pulled Auvi-Q following quality control issues. The device has since been put back on the market by another pharmaceutical company, Kaléo. The list price of the newly released Auvi-Q is set at $4,500.

Mylan did not immediately respond to Ars' request for comment.

http://bit.ly/2qkY3As

Newly prescribed sleeping pills increase risk of hip fracture

Over double the odds of a hip fracture in the first two weeks compared with non-users

Older people newly prescribed sleeping pills like benzodiazepines and 'Z-drugs' have over double the odds of a hip fracture in the first two weeks compared with non-users, according to a new study by researchers at Cardiff University and King's College London.

Dr Ben Carter, Cardiff University's School of Medicine and the Institute of Psychiatry, Psychology and Neuroscience, King's College London, explains: "While 'Z-drugs are fast becoming the doctor's hypnotic prescription of choice, there is no evidence that they are a safer alternative to benzodiazepines in relation to hip fracture risk. "Our study shows that both appear to significantly increase the risk of hip fracture when newly prescribed by doctors."

The study of people aged over 65 found that new users of these hypnotic medicines experienced nearly two and a half times the fracture rate, when compared with older people not taking hypnotics. An estimated 53% increase in fracture risk was identified in medium-term users (15 to 30 days), as well as a 20% increased risk of hip fracture in long-term users (greater than 30 days).

Dr Carter added: "Careful consideration of the immediate increased risk of hip fracture should inform the clinical decision-making process. Clinically effective measures like strength training to improve frailty, removal of hazards at home, visual correction and a medication review are also needed to mitigate the risk of hip fractures, particularly in the first few days of use."

The research supports previous studies linking use of hypnotics by older people with an increased risk of accidents, dependence, cognitive decline and hip fracture. The drugs are also thought to cause drowsiness, delayed reaction times and impaired balance.

'Benzodiazepines, Z-drugs and the risk of hip fracture: A Systematic Review and Meta-Analysis' is published today in PLOS ONE.

http://bit.ly/2piYwVj

Drugs already in medicine cabinets may fight dementia, early data suggests

In mouse and cell studies, drugs shut down damaging stress response, protected brain.

Beth Mole - 4/26/2017, 2:44 AM

Tried, true, and FDA-approved drugs for cancer and depression—already in medicine cabinets—may also be long-sought treatments for devastating brain diseases such as Alzheimer's, Parkinson's, and other forms of dementia, according to a new study in *Brain, a Journal of Neurology*.

The research is still in early stages; it only involved mouse and cell experiments, which are frequently not predictive of how things will go

in humans. Nevertheless, the preliminary findings are strong, and scientists are optimistic that the drugs could one day help patients with progressive brain disease. Researchers are moving toward human trials. And this process would be streamlined because the drugs have already cleared safety tests. But even if the early findings hold up, it would still take years to reach patients.

In the preliminary tests, the two drugs—trazodone hydrochloride, used to treat depression and anxiety, and dibenzoylmethane (DBM), effective against prostate and breast tumors—could shut down a devastating stress response in brain cells, known to be critical for the progression of brain diseases. The drugs both protected brain cells and restored memory in mice suffering from brain diseases.

"We're excited by the potential of these findings from this well-conducted and robust study," Doug Brown, of the Alzheimer's Society, told the *BBC*.

David Dexter, from Parkinson's UK, added that "if these studies were replicated in human clinical trials, both trazodone and DBM could represent a major step forward."

Stressful stress response

For years, researchers have known that a stress response in cells, called "unfolded protein response," or UPR, is involved in a bunch of neurodegenerative diseases. The response kicks in when there's a buildup of unfolded or misfolded proteins. Typically, protein chains are folded into specific 3D structures that are often critical for their function in the body. But this folding goes awry in some neurodegenerative conditions, such as prion diseases, Alzheimer's disease, Parkinson's disease, and other forms of dementia.

When this happens, UPR kicks in. It shuts down protein production, tries to junk the botched proteins, and gets protein production machinery back in order. If all goes well, the cell can resume normal protein production. But if it doesn't, UPR initiates apoptosis, aka cell suicide.

In neurodegenerative diseases, things don't go well; UPR is over-activated, and brain cells start dying off. Scientists know that hampering UPR can protect brain cells and restore memory in mice engineered to mimic having Alzheimer's disease. But so far, all the compounds found to knock back UPR were highly toxic or highly insoluble (they don't work as medicine).

Cut to the drugs

For a drug discovery shortcut, researchers at the University of Cambridge wondered: do we already have drugs that can interfere with UPR but just don't know it? They screened a library of 1,040 FDA-approved drugs to find out.

Because UPR is highly conserved across animals, the researchers could use worms to screen the drugs. They initially found 20 drugs that seemed to have UPR-dampening effects. Upon further testing, they whittled down the list to five, then to two.

In further cell experiments, both trazodone and DBM inhibited a specific step in UPR and restored protein production. In mice, the drugs traversed the blood-brain barrier. When the researchers infected mice with a prion disease, clinically relevant doses of either drug held back neurological symptoms, boosted survival, and substantially reduced loss of brain cells in most of the infected mice. In mice that modeled a type of dementia, called frontotemporal dementia, both drugs could rescue the rodents' memory and restore protein synthesis in brain cells.

"The two drugs were markedly neuroprotective," the authors conclude. "These drugs therefore represent an important step forward in the pursuit of disease-modifying treatments for Alzheimer's and related disorders."

Trazodone, the authors note, is even already approved for use in the elderly. Clinical trials are the next step.

Brain, *2017. DOI: 10.1093/brain/awx074 (About DOIs).*

http://go.nature.com/2pL0LlI

More surgeons must start doing basic science

They say they don't have the time or incentives to do research — and that's dangerous for translational medicine.

In Steven Soderbergh's classy television show *The Knick*, set in a New York City hospital in the early 1900s, competitive and obsessively driven surgeon-scientists work on the burning medical issues of the day — identification of blood groups to allow blood transfusions, for example, and facial reconstruction surgery that returns dignity to those disfigured by syphilis.

Would-be healers have been testing surgical procedures since the Iron Age first delivered the necessary cutting tools. And the need for surgical advances remains. From the first heart transplant in 1967 to the emergence of deep brain stimulation and hopes for regenerative medicine, research is needed to transfer benchside discoveries to the bedside.

It is a problem, then, to find that surgeons are increasingly turning their backs on research. Evidence suggests that, compared with a decade or two ago, surgeons apply for and receive fewer grants, publish less, and - perhaps most perniciously - feel that research is not part of their role. Anecdotal reports suggest the trend is widespread, and not restricted to the United States - where it is best documented.

The latest report on the subject, published last September in *Annals of Surgery*, indicates that, according to two different measures, academic surgeons' interest in research in the United States is falling in linear fashion (S. G. Keswani *et al. Ann. Surg.* http://doi.org/b52r; 2016).

The report, compiled by the Society of University Surgeons (SUS), looked at grants awarded by the National Institutes of Health (NIH) to the 25 top-funded academic medical centres, and found that the proportion of funding to surgical departments dropped from 3% to 2.3% over the period 2006–14. Within individual medical faculties, the proportion of money going to surgical departments also fell. This is consistent with earlier studies showing that fewer surgeon-scientists apply for NIH grants and that those who do tend to be less successful than their medical colleagues in non-surgical disciplines (S. J. Rangel and R. L. Moss *Surgery* **136,** 232–239; 2004).

The SUS report also looked at the number of abstracts submitted to the annual Academic Surgical Congress between 2011 and 2015, and found that the proportion relating to basic science fell by 24%.

What is behind this dismaying trend? In a survey conducted among academic surgeons in 2000, the majority of respondents reported a belief in the value of basic scientific research, even if they were finding that growing clinical and administrative duties hindered their success. But by the time of the SUS report, there had been a mood shift. Some 1,000 academic surgeons responded to a survey that the authors carried out. More than half said that basic research was a priority in their departments — but just one-third said that it was realistic to expect surgeons to succeed in basic research. Most respondents said they had neither the time nor the motivation for research, and in any case lacked adequate departmental support and funding. Nearly two-thirds believed that basic research among trainees should be limited to a select few residents with the ambition and talent to be successful in future research activities.

Non-surgical medical departments are not affected in the same way. This is probably because the time pressures on surgeons are even greater than those on other physicians. Surgeons are faced with the same increases in administrative duties as other medical-faculty members, but their clinical duties have grown faster. US hospitals depend increasingly on the income that surgeons generate — and have little motivation for encouraging them to spend time on research.

The flow of surgeons out of research is a problem that must be recognized and stopped. Translational medicine needs them too much. Transplantation and transplant immunology have always been dominated by surgeons, and these areas are set to embrace a future that includes regenerative medicine and possibly xenotransplantation (transplantation of tissues and organs from other species). They are

also much needed for crucial research into surgically treated diseases that only rarely hit the headlines — particularly in the correction of congenital birth defects, but also in adult disorders that rely on surgical skills, such as pancreatic cancers.

Involvement in research also allows surgeons to develop rigour in their everyday work, and to judge - and so maintain and improve - the quality of the work done by their peers.

Policymakers must create a health-care environment in which hospitals have incentives to think of patient care as inevitably linked to science, and to stop seeing surgeons as easy sources of revenue. But that's not going to happen any time soon. In the meantime, and at the very least, funding agencies should make it less burdensome for busy surgeons to apply for grants — and, in response, academic surgeons should apply more often, and thus increase their chances of success.

http://bit.ly/2qlf1bf

Humans Mastered Advanced Weapon-Making Technique 77,000 Years Ago

The discovery of 25 dangerously pointy stone weapons in a South African cave shows that humans mastered a complex weapon-creating technique during the Stone Age, some 77,000 years ago, according to a new study.

By Laura Geggel, Senior Writer | April 26, 2017 02:03pm ET

The discovery is the earliest evidence on record of a technique known as "pressure flaking," the researchers said. The technique is performed by using a pointed bone tool to remove small flakes of rock from a sharpened stone, the scientists said.

In contrast with other stone-flaking techniques, pressure flaking gives people more control over how to fashion and refine the sharp edges of a weapon, said study lead researcher Veerle Rots, a research professor at the Fund for Scientific Research at the University of Liège in Belgium. [In Photos: The Clovis Culture & Stone Tools]

Researchers unearthed the stone weapons in 2013 and 2014 in Sibudu, a cave located about 9 miles (15 kilometers) from South Africa's coast on the Indian Ocean. The weapons date to the Middle Stone Age in South Africa (a period starting about 300,000 years ago), a time known for its technological advancements, the researchers said. During this period, hunter-gatherer groups began using manipulative methods, such as heat and pressure, to produce stone weapons.

Of the 25 sharpened stone weapons, researchers analyzed this group of 14 that have serrated edges. Credit: Rots V. et al./PLOS ONE

The research team noted that archaeologists have found evidence of pressure flaking elsewhere. For instance, 75,000-year-old stone weapons manufactured with pressure flaking were found in Blombos Cave in South Africa. In Europe, pressure flaking is much younger, about 25,000 to 20,000 years old, Rots said.

However, it's anyone's guess exactly when and where these techniques were developed, the researchers said. The recent findings help scientists get closer to that answer, they said.

Weapon analysis

After finding the weapons, the researchers analyzed them in different ways, including by looking at their organic residues and wear and tear, as well as by experimentally reproducing them.

A handful of the weapons had two faces, which were likely produced by applying pressure to both sides of the stone, the researchers said. In some cases, these bifacial stones were attached to wooden shafts with a sticky resin, possibly to transform the stone points into weapons that

could be thrown from a distance, such as on a spear or an arrow, the researchers said.

Of the 25 stone weapons uncovered in the cave, 14 had evidence of impact-related damage, animal residues, and wear and tear, indicating that these stones were used for hunting, Rots added.

Some of the organic residues were more than 77,000 years old and had "evidence of glue, animal residues, including blood and bone fragments, [and] plant residues, including fibers," Rots said.

In all, the discovery is evidence that "specialized hunting technology was used in South Africa before 77,000 years ago," Rots told Live Science in an email.

The study was published online today (April 26) in the journal PLOS ONE.

http://bit.ly/2pxv6mX

National mental-health survey finds widespread ignorance, stigma

Less than half of Americans can recognize anxiety. Most people don't know what to do about depression even when they spot it. And nearly 8 in 10 don't recognize prescription drug abuse as a treatable problem.

EAST LANSING, Mich. - Those are just some of the findings of a new national survey on issues surrounding mental-health literacy by Michigan State University scholars. Their federally funded research comes as public health officials and advocates prepare to observe Mental Health Month in May.

"Our work is designed to help communities think about how to address behavioral health challenges as they emerge, whether that's drug abuse, anxiety or other issues, and the challenges such as suicide that can accompany them," said Mark Skidmore, an MSU professor and co-investigator on the project.

The national survey examines mental health literacy on four major issues: anxiety, depression, alcohol abuse and prescription drug abuse. The work is funded by the U.S. Department of Agriculture and the Substance Abuse and Mental Health Services Administration, an agency within the U.S. Department of Health and Human Services, and administered by the National Institute of Food and Agriculture, within Agriculture Department.

Skidmore said the web-based survey - which involved nearly 4,600 total participants - aims to give health officials and policymakers a better understanding of where to target education and prevention efforts for major societal issues such as prescription drug abuse. Public health officials are calling the opioid epidemic - which killed more than 33,000 people in 2015 - the worst drug crisis in American history.

According to the survey, 32 percent of all respondents were unable to identify the signs of prescription drug abuse (taking higher doses than prescribed, excessive mood swings, changes in sleeping patterns, poor decision-making and seeking prescriptions from more than one doctor). Those percentages were even more concerning for people aged 18-34 (47 percent) and among all men (44 percent).

"Although great strides have been made in the area of mental health literacy in recent decades," the authors write, "the discrepancies in mental health knowledge, helping behaviors and stigma show the importance of continuing to educate the public about mental health issues."

http://bbc.in/2oLVV7U

'Exciting' blood test spots cancer a year early

Doctors have spotted cancer coming back up to a year before normal scans in an "exciting" discovery.

By James Gallagher Health and science reporter, BBC News website

The UK team was able to scour the blood for signs of cancer while it was just a tiny cluster of cells invisible to X-ray or CT scans. It should allow doctors to hit the tumour earlier and increase the chances of a cure. They also have new ideas for drugs after finding how unstable DNA fuels rampant cancer development.

The research project was on lung cancer, but the processes studied are so fundamental that they should apply across all cancer types. Lung cancer kills more people than any other type of tumour and the point of the study is to track how it can "evolve" into a killer that spreads through the body.

Blood test

In order to test for cancer coming back, doctors need to know what to look for. In the trial, funded by Cancer Research UK, samples were taken from the lung tumour when it was removed during surgery.

A team at the Francis Crick Institute, in London, then analysed the tumour's defective DNA to build up a genetic fingerprint of each patient's cancer. Then blood tests were taken every three months after the surgery to see if tiny traces of cancer DNA re-emerged.

The results, outlined in the journal Nature, showed cancer recurrence could be detected up to a year before any other method available to medicine. The tumours are thought to have a volume of just 0.3 cubic millimetres when the blood test catches them.

'New hope'

Dr Christopher Abbosh, from the UCL Cancer Institute, said: "We can identify patients to treat even if they have no clinical signs of disease, and also monitor how well therapies are working. "This represents new hope for combating lung cancer relapse following surgery, which occurs in up to half of all patients."

So far, it has been an early warning system for 13 out of 14 patients whose illness recurred, as well as giving others an all-clear. In theory, it should be easier to kill the cancer while it is still tiny rather than after it has grown and become visible again. However, this needs testing.

Prof Charles Swanton, from the Francis Crick Institute, told the BBC: "We can now set up clinical trials to ask the fundamental question - if you treat people's disease when there's no evidence of cancer on a CT scan or a chest X-ray can we increase the cure rate?

"We hope that by treating the disease when there are very few cells in the body that we'll be able to increase the chance of curing a patient."

Janet Maitland, 65, from London, is one of the patients taking part in the trial. She has watched lung cancer take the life of her husband and was diagnosed herself last year. She told the BBC: "It was my worst nightmare getting lung cancer, and it was like my worse nightmare came true, so I was devastated and terrified."

But she had the cancer removed and now doctors say she has a 75% chance of being cancer-free in five years. "It's like going from terror to joy, from thinking that I was never going to get better to feeling like a miracle's been acted," she said. And taking part in a trial that should improve the chances for patients in the future is a huge comfort for her. "I feel very privileged," she added.

Evolution

The blood test is actually the second breakthrough in the massive project to deepen understanding of lung cancer.

A bigger analysis, published in the New England Journal of Medicine, showed the key factor - genetic instability - that predicted whether the cancer would return.

Multiple samples from 100 patients containing 4.5 trillion base pairs of DNA were analysed. DNA is packaged up into sets of chromosomes containing thousands of genetic instructions.

The team at the Francis Crick Institute showed tumours with more "chromosomal chaos" - the ability to readily reshuffle large amounts of their DNA to alter thousands of genetic instructions - were those most likely to come back.

Prof Charles Swanton, one of the researchers, told the BBC News website: "You've got a system in place where a cancer cell can alter its behaviour very rapidly by gaining or losing whole chromosomes or parts of chromosomes. "It is evolution on steroids."

That allows the tumour to develop resistance to drugs, the ability to hide from the immune system or the skills to move to other tissues in the body.

'Exciting'

The first implication of the research is for drug development - by understanding the key role of chromosomal instability, scientists can find ways to stop it.

Prof Swanton told me: "I hope we'll be able to generate new approaches to limit it and bring evolution back from the brink, perhaps reduce the evolutionary capacity of tumours and hopefully stop them adapting. "It's exciting on multiple levels."

The scientists say they are only scratching the surface of what can be achieved by analysing the DNA of cancers.

http://bit.ly/2pL7AUG

The upside of worrying

New study by UC Riverside psychology professor shows there's a positive side to worrying

RIVERSIDE, Calif. -- Worry - it does a body good. And, the mind as well. A new paper by Kate Sweeny, psychology professor at the University of California, Riverside, argues there's an upside to worrying.

"Despite its negative reputation, not all worry is destructive or even futile," Sweeny said. "It has motivational benefits, and it acts as an emotional buffer."

In her latest article, "The Surprising Upsides of Worry," published in Social and Personality Psychology Compass, Sweeny breaks down the role of worry in motivating preventive and protective behavior, and how it leads people to avoid unpleasant events. Sweeny finds worry is associated with recovery from traumatic events, adaptive preparation and planning, recovery from depression, and partaking in activities that promote health, and prevent illness. Furthermore, people who report greater worry may perform better -- in school or at the workplace -- seek more information in response to stressful events, and engage in more successful problem solving.

Worry as a Motivator

The motivational power of worry has been studied and linked to preventive health behavior, like seatbelt use. In a nationally representative sample of Americans, feelings of worry about skin cancer predicted sunscreen use. And participants who reported higher levels of cancer-related worries also conducted breast self-examinations, underwent regular mammograms, and sought clinical breast examinations.

"Interestingly enough, there are examples of a more nuanced relationship between worry and preventive behavior as well," Sweeny said. "Women who reported moderate amounts of worry, compared to women reporting relatively low or high levels of worry, are more likely to get screened for cancer. It seems that both too much and too little worry can interfere with motivation, but the right amount of worry can motivate without paralyzing."

In the paper, Sweeny noted three explanations for worry's motivating effects.

1. Worry serves as a cue that the situation is serious and requires action. People use their emotions as a source of information when making judgements and decisions.

2. Worrying about a stressor keeps the stressor at the front of one's mind and prompts people toward action.

3. The unpleasant feeling of worry motivates people to find ways to reduce their worry.

"Even in circumstances when efforts to prevent undesirable outcomes are futile, worry can motivate proactive efforts to assemble a ready-made set of responses in the case of bad news," Sweeny said. "In this instance, worrying pays off because one is actively thinking of a 'plan B.'"

Worry as a Buffer

Worry can also benefit one's emotional state by serving as an emotional bench-mark. Compared to the state of worry, any other feeling is pleasurable by contrast. In other words, the pleasure that comes from a good experience is heightened if preceded by a bad experience.

"If people's feelings of worry over a future outcome are sufficiently intense and unpleasant, their emotional response to the outcome they

ultimately experience will seem more pleasurable in comparison to their previous, worried state," Sweeny said.

Research on bracing for the worst provides indirect evidence for the role of worry as an emotional buffer, according to Sweeny. As people brace for the worst, they embrace a pessimistic outlook to mitigate potential disappointment, boosting excitement if the news is good. Therefore, both bracing and worrying have an emotional payoff following the moment of truth.

"Extreme levels of worry are harmful to one's health. I do not intend to advocate for excessive worrying. Instead, I hope to provide reassurance to the helpless worrier - planning and preventive action is not a bad thing," Sweeny said. "Worrying the right amount is far better than not worrying at all."

http://bit.ly/2puuMU1

Long After Their Bones Were Gone, Neanderthals' DNA Survived in a Cave

DNA from two extinct human relatives — the Neanderthals, and a mysterious branch of humanity called the Denisovans — has been detected in the ancient mud of caves, even though those caves hold no fossils of those individuals, new research shows.

By Charles Q. Choi, Live Science Contributor

The finding suggests that scientists could detect such extinct lineages in places devoid of skeletal remains, the researchers said. This technique, if verified, could fill blank spots in scientists' understanding of how and where humans evolved, according to the authors of the new study describing the finding.

Human remains are scarce

The ancestors of modern humans once shared the world with archaic human lineages such as the Neanderthals — the closest extinct relatives of modern humans — as well as the Denisovans. Little is known about the Denisovans, but scientists think this ancient human relative might have roamed a vast range stretching from Siberia to Southeast Asia. DNA extracted from fossilized bones and teeth of Neanderthals and Denisovans has revealed many secrets about human evolution, such as how modern humans interbred with both lineages.

But although there are numerous prehistoric sites that hold tools and other artifacts from ancient humans — such as the ancestors of modern humans, or members of extinct human lineages — their skeletal remains are scarce, thus limiting research into human evolution. Moreover, the ancient human fossils that archaeologists do unearth do not always have enough suitable DNA for genetic analysis.

Scientists found DNA related to the extinct human lineage called Denisovans in Denisova Cave in Siberia. Here, Richard (Bert) Roberts, Vladimir Ulianov and Maxim Kozlikin (clockwise from top) plan the sampling of sediments in the cave's east chamber. **IAET SB RAS/Sergei Zelensky**

"Humans are a very small proportion of the fauna found in caves," said study senior author Matthias Meyer, a geneticist at the Max Planck Institute for Evolutionary Anthropology in Leipzig, Germany. "In most excavation sites, if you find thousands of bones from animals, you're very lucky if you find one human tooth or a long-bone fragment."

No bones, no problem

Instead, Meyer and his colleagues investigated whether ancient sediments found in caves might latch on to DNA. "We know that with DNA preserved in bones, the DNA binds to the mineral component of bone, so the same can, in principle, happen in sediments full of minerals," Meyer said.

The scientists collected 85 samples of sediment covering a time span from 14,000 to more than 550,000 years ago, from seven sites in Belgium, France, Spain, Croatia and Russia, where previous research suggested ancient humans once lived. These sites included Denisova

Cave in Siberia, which is where Denisovan fossils were first discovered.

The researchers identified DNA from a variety of mammals, including woolly mammoths, woolly rhinoceroses, cave bears and cave hyenas. Mixed in with this animal DNA were small traces of human DNA: The researchers found Neanderthal DNA in four caves, and Denisovan DNA in Denisova Cave.

"The fact that sediment can indeed preserve DNA from extinct humans that lived there thousands of years ago is a pretty amazing finding," Meyer said.

In addition, at each of the two sites where the researchers did not discover DNA from ancient humans, they had only a few samples to analyze, Meyer noted. "Maybe if we looked at more samples from each site, we'd find Neanderthal or Denisovan DNA as well," he said.

DNA potential

The scientists aren't sure what part of the bodies of the extinct human lineages this DNA came from — for instance, skin flakes, hairs or bodily fluids such as sweat or blood. "Another possibility stems from how, in many sites, we find a lot of hyena DNA," Meyer said. "Maybe the hyenas were eating human corpses outside the caves, and went into the caves and left feces there, and maybe entrapped in the hyena feces was human DNA."

Scientists took samples from different layers of this sediment profile in Trou Al'Wesse cave in Belgium. They ran genetic analyses on the samples. **Monika V. Knul**

Most of the DNA from extinct humans that was recovered came from layers of sediment where no human fossils had been found previously. This suggests that, in the future, DNA could help researchers detect the presence of humans even in the absence of their skeletal remains, the study authors said.

For instance, "there are some very interesting open questions regarding the Denisovans — we only have fossils of them from a single site in Russia, but we know they must have been much more widespread due to the pattern of interbreeding we see with modern humans," Meyer said. "By looking for DNA, there's the chance we can find many more Denisovan sites than we would by just looking for bones or teeth."

One concern, however, is that DNA could seep across layers of sediment, thus making it difficult to figure out when, specifically, extinct humans or others lived at a site. (The deeper a layer of sediment is, the older it usually is.)

Still, the research team "didn't find any obvious evidence of DNA movement," Meyer said, "but it's certainly a possibility that needs to be investigated for every site."

Depending on how well DNA is preserved in any given cave, scientists "could learn much more information," Meyer added. "There's big potential here," he said, "and we need to do more work to understand just how big that potential is." The scientists detailed their findings online today (April 27) in the journal Science.

http://bit.ly/2qlM7yy

NASA study challenges long-held tsunami formation theory

A new NASA study is challenging a long-held theory that tsunamis form and acquire their energy mostly from vertical movement of the seafloor.

An undisputed fact was that most tsunamis result from a massive shifting of the seafloor—usually from the subduction, or sliding, of one tectonic plate under another during an earthquake. Experiments conducted in wave tanks in the 1970s demonstrated that vertical uplift of the tank bottom could generate tsunami-like waves. In the following decade, Japanese scientists simulated horizontal seafloor

displacements in a wave tank and observed that the resulting energy was negligible. This led to the current widely held view that vertical movement of the seafloor is the primary factor in tsunami generation.

In 2007, Tony Song, an oceanographer at NASA's Jet Propulsion Laboratory in Pasadena, California, cast doubt on that theory after analyzing the powerful 2004 Sumatra earthquake in the Indian Ocean. Seismograph and GPS data showed that the vertical uplift of the seafloor did not produce enough energy to create a tsunami that powerful. But formulations by Song and his colleagues showed that once energy from the horizontal movement of the seafloor was factored in, all of the tsunami's energy was accounted for. Those results matched tsunami data collected from a trio of satellites -the NASA/Centre National d'Etudes Spatiales (CNES) Jason, the U.S. Navy's Geosat Follow-on and the European Space Agency's Environmental Satellite.

Further research by Song on the 2004 Sumatra earthquake, using satellite data from the NASA/German Aerospace Center Gravity Recovery and Climate Experiment (GRACE) mission, also backed up his claim that the amount of energy created by the vertical uplift of the seafloor alone was insufficient for a tsunami of that size.

"I had all this evidence that contradicted the conventional theory, but I needed more proof," Song said.

His search for more proof rested on physics—namely, the fact that horizontal seafloor movement creates kinetic energy, which is proportional to the depth of the ocean and the speed of the seafloor's movement. After critically evaluating the wave tank experiments of the 1980s, Song found that the tanks used did not accurately represent either of these two variables. They were too shallow to reproduce the actual ratio between ocean depth and seafloor movement that exists in a tsunami, and the wall in the tank that simulated the horizontal seafloor movement moved too slowly to replicate the actual speed at which a tectonic plate moves during an earthquake.

"I began to consider that those two misrepresentations were responsible for the long-accepted but misleading conclusion that horizontal movement produces only a small amount of kinetic energy," Song said.

Building a Better Wave Tank

To put his theory to the test, Song and researchers from Oregon State University in Corvallis simulated the 2004 Sumatra and 2011 Tohoku earthquakes at the university's Wave Research Laboratory by using both directly measured and satellite observations as reference. Like the experiments of the 1980s, they mimicked horizontal land displacement in two different tanks by moving a vertical wall in the tank against water, but they used a piston-powered wave maker capable of generating faster speeds. They also better accounted for the ratio of how deep the water is to the amount of horizontal displacement in actual tsunamis.

The new experiments illustrated that horizontal seafloor displacement contributed more than half the energy that generated the 2004 and 2011 tsunamis.

"From this study, we've demonstrated that we need to look at not only the vertical but also the horizontal movement of the seafloor to derive the total energy transferred to the ocean and predict a tsunami," said Solomon Yim, a professor of civil and construction engineering at Oregon State University and a co-author on the study.

The finding further validates an approach developed by Song and his colleagues that uses GPS technology to detect a tsunami's size and strength for early warnings.

The JPL-managed Global Differential Global Positioning System (GDGPS) is a very accurate real-time GPS processing system that can measure seafloor movement during an earthquake. As the land shifts, ground receiver stations nearer to the epicenter also shift. The stations can detect their movement every second through real-time communication with a constellation of satellites to estimate the amount and direction of horizontal and vertical land displacement that

took place in the ocean. They developed computer models to incorporate that data with ocean floor topography and other information to calculate the size and direction of a tsunami.

"By identifying the important role of the horizontal motion of the seafloor, our GPS approach directly estimates the energy transferred by an earthquake to the ocean," Song said. "Our goal is to detect a tsunami's size before it even forms, for early warnings."

The study is published in Journal of Geophysical Research—Oceans.

http://bit.ly/2qinMML

Scientific papers that use old and new knowledge get the most attention

Top 5% of the most cited research papers draw upon a mix of old and new knowledge

April 27, 2017 by Laurel Thomas Gnagey

An examination of millions of scientific papers and patents reveals works that land in the top 5 percent of the most cited research draw upon a mix of old and new knowledge—significant in a day and age when the number of new publications is increasing dramatically, says a researcher at the University of Michigan.

Daniel Romero, assistant professor at the U-M School of Information, and colleagues from Northwestern University analyzed more than 28.4 million scientific papers in the Web of Science and nearly 5.4 million U.S. patents to determine which works were cited the most and why. They found what they referred to as the hotspot in which researchers doubled their chance of being cited.

This hotspot occurred when the research used a low mean age and high age variance, in other words when it included the latest information available in combination with some tried-and-true research from the past.

"It is natural for scientists to gravitate towards popular and 'hot' research topics and ideas," Romero said. "While focusing on solving recent problems and being aware of the state-of-the-art is crucial to make impact, we find that this is only half of the story. Old and established knowledge is also important and should be part of our thinking when we do research."

The authors note the vast acceleration in the production of new knowledge and the tendency for researchers to think newer is better. On the Web of Science, 1.5 million new articles were published in 2014 alone, sextupling since 1970, while the U.S. issued 287,831 patents in 2013, quadruple the number in 1970.

The researchers found these patterns to be true across science and technology fields. They also found that research involving collaborations was more likely to land in the hotspot than the work done by a solo researcher, which they say is not surprising since a team is likely to bring broader, more diverse knowledge to a problem than one individual.

"Our results have significance to the way researchers approach the search for ideas to draw on when they formulate their research," Romero said "Our results also have implications for academic search engines. Search engines could take our findings into account when developing ranking algorithms to avoid over-ranking recent works and providing users a healthy mixture of new and old papers."

More information: Satyam Mukherjee et al. The nearly universal link between the age of past knowledge and tomorrow's breakthroughs in science and technology: The hotspot, Science Advances (2017). DOI: 10.1126/sciadv.1601315

http://bit.ly/2pjzztc

How Humble Moss Healed the Wounds of Thousands in World War I

The same extraordinary properties that make this plant an "ecosystem engineer" also helped save human lives

By Lorraine Boissoneault

The First World War had just begun, and already the wounds were rotting on the battlefield. In the last months of 1914, doctors like Sir. W. Watson Cheyne of the Royal College of Surgeons of England noted with horror the "great prevalence of sepsis," the potentially life-threatening response triggered by a bad infection. And by December

1915, a British report warned that the thousands of wounded men were threatening to exhaust the material for bandages.

Desperate to get their hands on something sterile that would keep wounds clear of infection, doctors started getting creative. They tried everything from irrigating the wounds with chlorine solutions to creating bandages infused with carbolic acid, formaldehyde or mercury chloride, with varying degrees of success. But in the end, there simply wasn't enough cotton—a substance that was already in high demand for uniforms and its recently discovered use as an explosive—to go around.

What were the Allied Powers to do? A Scottish surgeon-and-botanist duo had an idea: stuff the wounds full of moss.

Yes, moss, the plant. Also known as sphagnum, peat moss thrives in cold, damp climates like those of the British Isles and northern Germany. Today, this tiny, star-shaped plant is known for its use in horticulture and biofuel, not to mention its starring role in preserving thousands-year-old "bog bodies" like the Tollund Man, which *Smithsonian Magazine* revisited last month. But humans have also used it for at least 1,000 years to help heal their injuries. …

In ancient times, Gaelic-Irish sources wrote that warriors in the battle of Clontarf used moss to pack their wounds. Moss was also used by Native Americans, who lined their children's cradles and carriers with it as a type of natural diaper. It continued to be used sporadically when battles erupted, including during the Napoleonic and Franco-Prussian wars. But it wasn't until World War I that medical experts realized the plant's full potential.

In the war's early days, eminent botanist Isaac Bayley Balfour and military surgeon Charles Walker Cathcart identified two species in particular that worked best for staunching bleeding and helping wounds heal: *S. papillosum* and *S. palustre*, both of which grew in abundance across Scotland, Ireland and England. When the men wrote an article in the "Science and Nature" section of *The Scotsman* extolling the moss's medicinal virtues, they noted that it was already widely used in Germany.

But desperate times called for desperate measures. Or, as they wrote: "Fas est et ab hoste doceri"—it is right to be taught even by the enemy. Field surgeons seemed to agree. Lieutenant-Colonel E.P. Sewell of the General Hospital in Alexandria, Egypt wrote approvingly that, "It is very absorbent, far more than cotton wool, and has remarkable deodorizing power." Lab experiments around the same time vindicated his observations: Sphagnum moss can hold up to 22 times its own weight in liquid, making it twice as absorptive as cotton.

This remarkable spongelike quality comes from Sphagnum's cellular structure, says Robin Kimmerer, professor of ecology at SUNY-Environmental Science and Forestry and the author of *Gathering Moss: A Natural and Cultural History of Mosses*. "Ninety percent of the cells in a sphagnum plant are dead," Kimmerer says. "And they're supposed to be dead. They're made to be empty so they can be filled with water." In this case, humans took advantage of that liquid-absorbing capacity to soak up blood, pus and other bodily fluids.

Sphagnum moss also has antiseptic properties. The plant's cell walls are composed of special sugar molecules that "create an electrochemical halo around all of the cells, and the cell walls end up being negatively charged," Kimmerer says. "Those negative charges mean that positively charged nutrient ions [like potassium, sodium and calcium] are going to be attracted to the sphagnum." As the moss soaks up all the negatively charged nutrients in the soil, it releases positively charged ions (protons) that make the environment around it acidic.

For bogs, the acidity has remarkable preservative effects—think bog bodies—and keeps the environment limited to highly specialized species that can tolerate such harsh environments. For wounded humans, the result is that sphagnum bandages produce sterile environments by keeping the pH level around the wound low, and inhibiting the growth of bacteria.

As the war raged on, the number of bandages needed skyrocketed, and sphagnum moss provided the raw material for more and more of them. In 1916, the Canadian Red Cross Society in Ontario provided over 1 million dressings, nearly 2 million compresses and 1 million pads for wounded soldiers in Europe, using moss collected from British Columbia, Nova Scotia and other swampy, coastal regions. By 1918, 1 million dressings per month were being sent out of Britain to hospitals on continental Europe, in Egypt and even Mesopotamia.

A vial of dried Sphagnum that would've been used for making bandages in WWI. **National Museum of American History**

Communities around the United Kingdom and North America organized outings to collect moss so the demand for bandages could be met. "Moss drives" were announced in local papers, and volunteers included women of all ages and children. One organizer in the United Kingdom instructed volunteers to "fill the sacks only about three-quarter full, drag them to the nearest hard ground, and then dance on them to extract the larger percentage of water."

At Longshaw Lodge in Derbyshire, England, the nurses who tended convalescing soldiers trooped out to the damp grounds to collect moss for their wounds. And as botanist P.G. Ayres writes, sphagnum was just as popular on the other side of the battle lines. "Germany was more active than any of the Allies in utilizing Sphagnum … the bogs of north-eastern Germany and Bavaria provided seemingly inexhaustible supplies. Civilians and even Allied prisoners of war were conscripted to gather the moss."

Each country had its own method for making the bandages, with the British stations filling bags loosely while the American Red Cross provided precise instructions for how to layer the moss with nonabsorbent cotton and gauze. "[The British style] seems to have been looked down upon by the American Red Cross," says Rachel Anderson, a project assistant in the division of medicine and science at the National Museum of American History who studied the museum's collection of sphagnum bandages. "The criticism was that you were getting redistribution of the moss during shipment and use."

But everyone agreed one on thing: moss bandages worked. Their absorbency was remarkable. They didn't mildew. And from the Allies' perspective, they were a renewable resource that would grow back without much difficulty. "So long as the peat underneath [the living moss] was not disturbed, the peat is going to keep acting like a sponge, so it enables regrowth of Sphagnum," says Kimmerer. However, "I can imagine if there were bogs that people used very regularly for harvesting there could be a trampling effect."

So why aren't we still using moss bandages today? In part, because the immense amount of labor required to collect it, Anderson says (although manufacturers in the U.S. experimented with using the moss for sanitary napkins called Sfag-Na-Kins).

That's a good thing, because the real value of this plant goes far beyond bandages. Peatlands full of spaghnum and other mosses spend thousands of years accumulating carbon in their underground layers. If they defrost or dry out, we risk that carbon leaking out into the atmosphere. And while humans are no longer picking them for bandages, scientists fear that bogs and swamplands could be drained or negatively impacted by agriculture and industry, or the peat will be used for biofuel.

Besides their role in global climate change, peatlands are rich ecosystems in their own right, boasting rare species like carnivorous plants. "The same things that make sphagnum amazing for bandages are what enable it to be an ecosystem engineer, because it can create bogs," Kimmerer says. "Sphagnum and peatlands are really important pockets of biodiversity." Even if we no longer require moss's assistance with our scrapes and lacerations, we should still respect and preserve the rare habitats it creates.

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"Out of his mind" surgeon plans human head transplant, revival of frozen brain

They're preposterous claims.

Beth Mole - 4/29/2017, 2:19 AM

Italian neurosurgeon Sergio Canavero will undertake the first human head transplant later this year in China, the doctor told German magazine *Ooom* in an article published Thursday. And, following that effort, he will revive a cryogenically frozen brain and transplant it into a donor body within the next three years.

The plans, completely disconnected from reality and the state of modern medicine, are at least in line with his previous outlandish goals and dubious animal research.

Canavero made headlines in the past few years by claiming that transplanting the whole head of a human onto a donor body is currently possible. A Russian man, suffering from a spinal muscular atrophy malady called Werdnig-Hoffmann Disease, even publicly volunteered for the procedure.

As proof that the transplant could work, Canavero published gruesome experiments in 2016, said to have repaired the severely injured spinal cords of mice, rats, and a dog.

The experiments came complete with cringe-worthy video of recovering animals struggling to drag their limp bodies around. Yet, the study lacked controls, detailed methods, and data on the injuries and recoveries. Canavero claimed to perform a head transplant on a monkey but did not publish the experiment.

Experts decidedly consider his research on spinal cord repair, let alone whole head transplants, unconvincing. A medical ethicist dubbed Canavero "out of his mind" for sweeping past the currently insurmountable challenges of such feats. These include intricately repairing and reattaching thousands of delicate nerves and restoring function.

Right now, doctors can't even convince the immune system to accept far simpler transplants consistently. There's also the completely unknown effects of such a transplant on the powerful human psyche.

Canavero is carrying on, undeterred it seems. In his *Ooom* interview, he not only glided through the idea of successfully transplanting a head, he made an even more absurd claim: that he would revive a cryogenically frozen brain and transplant it into a donor body. Canavero said he would obtain a preserved brain from Alcor Life Extension Foundation, a cryonics company located in Scottsdale, Arizona, according to Gizmodo.

There is currently no way to revive and molecularly repair a frozen human brain. And such transplants haven't even been attempted in animals. Thus, the surgical procedure is decades if not centuries away.

As Gizmodo also reports, Alcor said that Canavera hadn't even contacted the company. It distanced itself from the doctor, as did other cryonics leaders, and noted that his efforts are not realistic or even a shared goal.

In a statement, the company said:

The Alcor Life Extension has had no contact with Dr. Canavero. It is not yet possible to revive human brains cryopreserved with present methods. Revival of today's cryonics patients will require future repair by highly advanced future technology, such as molecular nanotechnology.

Technology that is advanced enough to repair a cryopreserved brain would by its nature also be able to regrow new tissues, organs, and a healthy body for the revived person. Therefore Alcor does not expect body donations or transplants to ever be necessary for revival of cryonics patients.

Until advanced tissue regeneration technology is developed, we wish Dr. Canavero well in his development of body transplant surgery for living patients today who might benefit.

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Study finds bonobos may be better representation of the last common ancestor with humans than common chimpanzees

Firsthand evidence that bonobos may be more closely linked to human ancestors than chimpanzees

A new study examining the muscular system of bonobos provides firsthand evidence that the rare great ape species may be more closely linked to human ancestors than common chimpanzees.

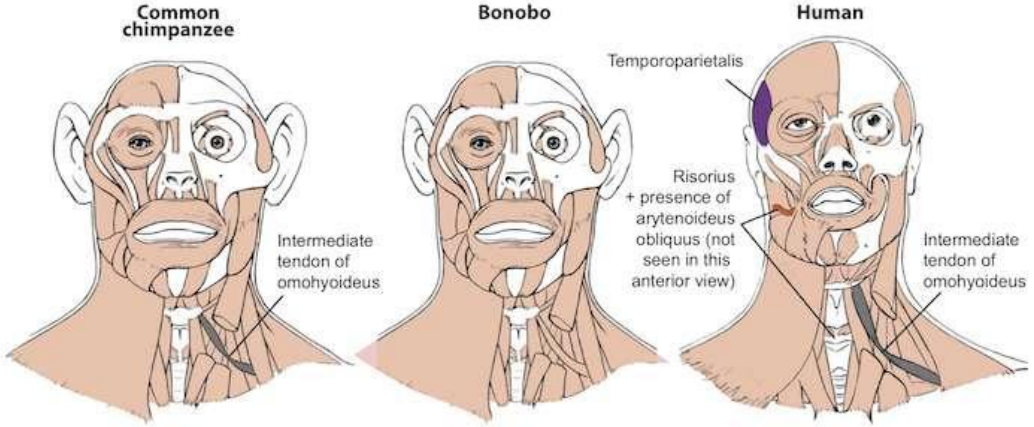
Credit: iStock

A new study examining the muscular system of bonobos provides firsthand evidence that the rare great ape species may be more closely linked, anatomically, to human ancestors than common chimpanzees. Previous research suggested this theory at the molecular level, but this is the first study to compare in detail the anatomy of the three species.

"Bonobo muscles have changed least, which means they are the closest we can get to having a 'living' ancestor," said Bernard Wood, professor of human origins at the GW Center for the Advanced Study of Human Paleobiology.

Scientists believe that modern human and common chimpanzee/bonobo lineages split about 8 million years ago with the two great ape species splitting about 2 million years ago. As common chimpanzees and bonobos evolved after their split, they developed different traits and physical characteristics, even though they remained geographically relatively close, with their main division being the Congo River. Because of this, researchers have been curious as to what those differences are and how they compare to humans. By studying the muscles of bonobos (which indicates how they physically function), the team was able to discover that they are more closely related to human anatomy than common chimpanzees, in the sense that their muscles have changed less than they have in common chimpanzees.

Earlier studies examined the DNA similarities and differences between bonobos and common chimpanzees, but this was the first study to compare the muscles of the three species.



These are differences between head muscles of common chimpanzees, bonobos and modern humans. There are no major consistent differences concerning the presence/absence of muscles in adult common chimpanzees (left) and bonobos (center), the only minor difference (shown in grey in the common chimpanzee scheme) being that the omohyoideus has no intermediate tendon in bonobos, contrary to common chimpanzees (and modern humans). In contrast, there are many differences between bonobos and modern humans (right) concerning the presence/absence of muscles in the normal phenotype (shown in colors and/or with labels in the human scheme). **Julia Molnar**

"In addition, our study has shown that there is a mosaic evolution of the three species, in the sense that some features are shared by humans and bonobos, others by humans and common chimpanzees, and still others by the two ape species," said Rui Diogo, lead author of the paper and associate professor of anatomy at Howard University. "Such a mosaic anatomical evolution may well be related to the somewhat similar molecular mosaic evolution between the three species revealed by previous genetic studies: each of the chimpanzees species share about 3 percent of genetic traits with humans that are not present in the other chimpanzee species."

The researchers led a team that examined seven bonobos from the Antwerp Zoo that had died and were being preserved. Researchers

said this was an extremely rare opportunity given bonobos' status as an endangered species.

The scientists note that having a clear understanding of what makes humans different from our closest living relatives might lead to new breakthroughs or understandings of human health.

The paper, "Bonobo anatomy reveals stasis and mosaicism in chimpanzee evolution, and supports bonobos as the most appropriate extant model for the common ancestor of chimpanzees and humans," published in *Scientific Reports*, a Nature publication, this month.

Explore further: *Genome sequencing reveals ancient interbreeding between chimpanzees and bonobos*

More information: ***Rui Diogo et al. Bonobo anatomy reveals stasis and mosaicism in chimpanzee evolution, and supports bonobos as the most appropriate extant mode l for the common ancestor of chimpanzees and humans, Scientific Reports (2017).***

http://bit.ly/2qkGpjh

Non-O blood groups associated with higher risk of heart attack

Having a non-O blood group is associated with a higher risk of heart attack

Paris, France - Having a non-O blood group is associated with a higher risk of heart attack, according to research presented today at Heart Failure 2017 and the 4th World Congress on Acute Heart Failure.1

Lead author Tessa Kole, a Master's degree student at the University Medical Centre Groningen, the Netherlands, said: "It has been suggested that people with non-O blood groups (A, B, AB) are at higher risk for heart attacks and overall cardiovascular mortality, but this suggestion comes from case-control studies which have a low level of evidence. If this was confirmed it could have important implications for personalised medicine."

The current study was a meta-analysis of prospective studies reporting on O and non-O blood groups, and incident cardiovascular events including myocardial infarction (heart attack), coronary artery disease, ischaemic heart disease, heart failure, cardiovascular events and cardiovascular mortality.

The study included 1 362 569 subjects from 11 prospective cohorts, described in nine articles. There were a total of 23 154 cardiovascular events. The researchers analysed the association between blood group and all coronary events, combined cardiovascular events, and fatal coronary events.

The analysis of all coronary events included 771 113 people with a non-O blood group and 519 743 people with an O blood group, of whom 11 437(1.5%) and 7 220 (1.4%) suffered a coronary event, respectively. The odds ratio (OR) for all coronary events was significantly higher in carriers of a non-O blood group, at 1.09 (95% confidence interval [CI] of 1.06-1.13).

The analysis of combined cardiovascular events included 708 276 people with a non-O blood group and 476 868 people with an O blood group, of whom 17 449 (2.5%) and 10 916 (2.3%) had an event, respectively. The OR for combined cardiovascular events was significantly higher in non-O blood group carriers, at 1.09 (95% CI 1.06-1.11).

The analysis of fatal coronary events did not show a significant difference between people with O and non-O blood groups.

"We demonstrate that having a non-O blood group is associated with a 9% increased risk of coronary events and a 9% increased risk of cardiovascular events, especially myocardial infarction," said Ms Kole.

The mechanisms that might explain this risk are under study. The higher risk for cardiovascular events in non-O blood group carriers may be due to having greater concentrations of von Willebrand factor, a blood clotting protein which has been associated with thrombotic events. Further, non-O blood group carriers, specifically those with an A blood group, are known to have higher cholesterol. And galectin-3, which is linked to inflammation and worse outcomes in heart failure patients, is also higher in those with a non-O blood group.

Ms Kole said: "More research is needed to identify the cause of the apparent increased cardiovascular risk in people with a non-O blood group. Obtaining more information about risk in each non-O blood

group (A, B, and AB) might provide further explanations of the causes."

She concluded: "In future, blood group should be considered in risk assessment for cardiovascular prevention, together with cholesterol, age, sex and systolic blood pressure. It could be that people with an A blood group should have a lower treatment threshold for dyslipidaemia or hypertension, for example. We need further studies to validate if the excess cardiovascular risk in non-O blood group carriers may be amenable to treatment."

http://bit.ly/2pAhtDB

Injecting virus into brain may relieve Parkinson's symptoms

Using a virus to reprogram cells in the brain could be a radical way to treat Parkinson's disease.

By Andy Coghlan

People with Parkinson's have difficulty controlling their movements due to the death of neurons that make dopamine, a brain signalling chemical. Transplants of fetal cells have shown promise for replacing these dead neurons in people with the disease, and a trial is currently under way.

But the transplant tissue comes from aborted pregnancies, meaning it is in short supply, and some people may find this ethically difficult. Recipients of these cells have to take immunosuppressant drugs too.

Ernest Arenas, at the Karolinska Institute in Stockholm, Sweden, and his team have found a new way to replace lost dopamine-making neurons. They injected a virus into the brains of mice whose dopamine neurons had been destroyed. This virus had been engineered to carry four genes for reprogramming astrocytes – the brain's support cells – into dopamine neurons.

Five weeks later, the team saw improvements in how the mice moved. "They walked better and their gait showed less asymmetry than controls," says Arenas. This is the first study to show that reprogramming cells in the living brain can lead to such improvements, he says.

Human cells

The effect of the virus was localised to the specific area where the team injected them. They did not see astrocytes turn into dopamine neurons in any other areas of the brain, nor were there any signs of tumours or other unwanted effects.

The team has also used the same four genes to convert human astrocytes into dopamine neurons in a dish, suggesting that a technique like this may be possible in people. However, Arenas says careful safety checks and improvements to the technique are necessary before such a procedure could be tried in people.

"The critical question will be whether this would work in the aged human brain, and generate enough dopamine cells of the right type that can connect up with the brain in the same way that transplanted dopamine cells can," says Roger Barker at the University of Cambridge, who is leading the fetal transplant trial.

Journal reference: Nature Biotechnology, DOI: 10.1038/nbt.3835

http://bit.ly/2oXZKSO

Novel antibacterial wound cover could prevent thousands of infections each year

Researchers develop bacteria-fighting wound dressing made with the help of crustaceans

Amsterdam - A new type of wound dressing could improve thousands of people's lives, by preventing them from developing infections. The dressing, a type of compression held in place by a bandage, uses an antibacterial substance formed from the shells of crustaceans like shrimps. It is described in a paper published in the May issue of Radiation Physics and Chemistry.

The innovative wound cover was made using a substance extracted from the shells of crustaceans

Antimicrobial resistance is becoming a worldwide health threat. A recent report by the Review on Antimicrobial Resistance,

commissioned in 2014 by then UK Prime Minister David Cameron and led by economist Jim O'Neill, warns that antimicrobial resistance could kill 10 million people each year by 2050, dwarfing even the number of estimated deaths from cancer. Because of this, preventing infection has never been more important.

The protective dressing was developed by Dr. Radoslaw Wach and his colleagues from Lodz University of Technology in Poland. Their innovation builds on a type of dressing that has been around for centuries. By providing moisture to a wound, hydrogel dressings can speed up aspects of healing and cool the wound down. The dressings are durable and elastic, meaning they can easily adapt to the shape of the affected body part.

Dr. Wach and his colleagues adapted the hydrogel dressing manufacturing technique to make a version of the classic dressing with an added benefit. The team did this by incorporating an antibacterial and biodegradable substance called chitosan, extracted from the shells of crustaceans, within the dressing itself.

The extraction process involves isolating a substance called chitin that is found in the shells and then changing its structure by removing most chemical branches from its acetyl groups. The resulting chitosan then has to be purified before it is used. Chitosan is useful in bandages to stop bleeding and has been known for its antimicrobial properties for decades.

Dr. Wach and his colleagues used a technique called irradiation to combine chitosan with hydrogel dressings. The method comprises cross-linking of hydrophilic polymers next to water -- just like with basic hydrogel dressings -- to form the firm and durable structure of the dressing and sterilize it in a single step. The researchers next shone an electron beam at the polymer containing a solution of chitosan in a substance called lactic acid while making the dressings. This allowed the chitosan to become part of the dressing itself.

"We developed a composition where chitosan is dissolved in lactic acid and, when added to the regular composition of the dressing, it does not adversely change its ability to cross-link during manufacturing or alter its mechanical and functional properties," said Dr. Wach. "The new hydrogel wound dressing is biologically active."

Dr. Wach hopes the new dressings will one day be used as a replacement for classic hydrogels. "Since wound healing in severe cases may take a long time -- up to several weeks -- the probability of bacteria-mediated infection is high," he added. "Our novel hydrogel dressing could, therefore, prevent many such infections and avoid serious complications."

Last month, the World Health Organization (WHO) published a new list of bacteria for which new antibiotics are needed. The most critical group of all includes bacteria resistant to multiple drugs. These pose a particular threat in hospitals, nursing homes, and among patients whose care requires devices such as ventilators and blood catheters. They include bacteria called Acinetobacter, Pseudomonas and various Enterobacteriaceae, including Klebsiella, E. coli, Serratia, and Proteus. Furthermore, 480,000 people develop multidrug-resistant tuberculosis globally each year, according to the WHO, and drug resistance is starting to complicate the fight against HIV and malaria. "If our solution is commercialized," concludes Dr. Walsh, "tens of thousands of infections could be prevented each year."

Notes for editors

The article is "Chitosan-containing hydrogel wound dressings prepared by radiation technique," by Wiktoria Mozalewska, Renata Czechowska-Biskup, Alicja K. Olejnik, Radoslaw A. Wach, Piotr Ula?ski and Janusz M. Rosiak. It appears in Radiation Physics and Chemistry, volume 134 (May 2017), published by Elsevier.