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The fish that have bellies full of mice – but we don't know how

It's a cat-and-mouse tale with a difference. The lesser salmon catfish has been found feasting on mice. But how does it catch them?

By Robin Wylie

Some catfish are known to ambush unwary pigeons at the water's edge, giving them the nickname "freshwater killer whales". But the lesser salmon catfish might just be an opportunist, gobbling up animals when they drown.

A survey of 18 lesser salmon catfish (Neoarius graeffei) from Ashburton river in northern Australia, suggests the fish can consume large quantities of small land animals when given the chance — almost half of the catfish had mice in their bellies.

"That is a lot, and a rare finding," says Peter Lisi, an aquatic ecologist at the University of Wisconsin-Madison.

The stomachs of some catfish contained as much as 95 per cent small mammals, with two fish having three animals each in their stomachs.

Lesser salmon catfish can grow to half a metre long and weigh up to 1.5 kilograms. They are a common species in dryland rivers of north-western Australia, so their diet is important to understanding the local ecosystems.

They were thought to feed mainly on aquatic invertebrates and plants, with the occasional addition of fruit and terrestrial insects, especially during the floods in the wet season.

And though a few freshwater fish species are known to dine on land vertebrates — African tigerfish have been filmed plucking a swallow out of thin air, for example — it is rare for them to eat so many.

The catfish had been mostly eating spinifex hopping mice (Notomys alexis, pictured above), which are around 10 centimetres long. As their name suggests, the mice get around by jumping. There are no reports of these mice intentionally spending any time in the water.

But heavy rain might have a role to play. "These mice often live in small colonies within a single burrow system," says Erin Kelly of the Centre for Fish and Fisheries Research at Murdoch University, Perth, who led the research, "so collapse or flooding of one or multiple burrow systems along the Ashburton river could have inadvertently introduced them into the water."

"When several catfish are targeting mice all at once, it suggests that a large pulse of mice are entering the river," says Lisi. "We still do not know how catfish gain access to mice or how often it occurs, or at what scale mice support river food webs. Because large fish often survive through feast and famine periods, big meals like this are ecologically relevant." If this is what is happening, the mice could be in greater danger as climate change kicks in.

"Climate projections for north-western Australia indicate that we're going to see both longer periods of drought and more intense rainfall events," Kelly says. "Changes in periods of flooding could possibly be altering the food web of these fish."

Journal reference: Journal of Arid Environments, DOI: 10.1016/j.jaridenv.2016.08.005

http://bit.ly/2c6uVGu

Did Lucy Die Falling From a Tree?

Researchers say they've figured a cause of death for the 3.2-million-year-old fossil, and it could change how we understand her daily life.

By Nathan Collins

It's been a mystery ever since they found the young woman's remains outside Hadar, Ethiopia, in 1974: How did Lucy, one of our earliest known ancestors, die?

Of course, it's not an easy task, figuring out a cause of death after 3.2 million years, but a new post-mortem suggests an answer: Most likely, she fell from a tree. It's an observation that adds to a long-standing debate over how it is we came to stand up and walk on two legs.

In case you need a quick refresher, Lucy is the nickname of a female Australopithecus afarensis, one of the earliest hominids to walk on two legs.

Despite her exalted status in the evolutionary history of humankind, researchers don't know a whole lot about how Lucy lived—in particular where she lived. Her long arms hint that she spent most of her time in trees, but her legs, clearly built for walking upright, suggest she may have spent more time on the ground. It's a matter of "vigorous debate," writes a team led by University of Texas–Austin anthropologist John Kappelman in Nature.

As is often the case, however, figuring out how Lucy died could reveal something about how she lived, so Kappelman and his team went back to the original skeleton, stored at the National Museum of Ethiopia.

Their analysis revealed a spiral fracture on Lucy's right humerus, or upper arm bone, in addition to evidence the head of the humerus had been crushed into the shaft of the bone, consistent with "an accident victim [who] consciously stretches out their arm in an attempt to break their fall," the team argues. What's more, the breaks are clean and show no signs of healing, indicating they occurred around the time of death.

"These humeral fractures were long thought to have occurred post-mortem, but their close match to clinical cases suggests instead that they represent perimortem injuries," Kappelman and his colleagues write.

Beyond curiosity about how Lucy died, the results add some credence to the hypothesis that she and her Australopithecus afarensis relatives may have lived in trees. The Hadar region was a mix of grasses and trees at the time, and modern chimpanzees live in tree nests, sometime at heights upwards of 100 feet, making it at least plausible that Lucy died in a fall from a tree she called home.

"Close inspection of other fossil specimens for antemortem or perimortem fractures … has the potential to offer important information about their lifestyles through an understanding of the trauma that they suffered and the mechanisms by which they died," the team writes.

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FSU research team makes Zika drug breakthrough

Discovery shows existing drugs can treat virus

TALLAHASSEE, Fla. -- A team of researchers from Florida State University, Johns Hopkins University and the National Institutes of Health has found existing drug compounds that can both stop Zika from replicating in the body and from damaging the crucial fetal brain cells that lead to birth defects in newborns. One of the drugs is already on the market as a treatment for tapeworm.

"We focused on compounds that have the shortest path to clinical use," said FSU Professor of Biological Science Hengli Tang. "This is a first step toward a therapeutic that can stop transmission of this disease."

Tang, along with Johns Hopkins Professors Guo-Li Ming and Hongjun Song and National Institutes of Health scientist Wei Zheng identified two different groups of compounds that could potentially be used to treat Zika -- one that stops the virus from replicating and the other that stops the virus from killing fetal brain cells, also called neuroprogenitor cells.

One of the identified compounds is the basis for a drug called Nicolsamide, a U.S. Food and Drug Administration approved drug that showed no danger to pregnant women in animal studies. It is commonly used to treat tapeworm. This could be prescribed by a doctor today, though tests are still needed to determine a specific treatment regimen for the infection. Their work is outlined in an article published Monday by Nature Medicine.

Though the Zika virus was discovered in 1947, there was little known about how it worked and its potential health implications -- especially among pregnant women -- until an outbreak occurred in South America last year. In the United States, there have been 529 cases of pregnant women contracting Zika, though most of those are travel related. As of Aug. 24, there have been 42 of locally transmitted cases in Florida. The virus, among other diseases, can cause microcephaly

in fetuses leading them to be born with severe birth defects. "It's so dramatic and irreversible," Tang said. "The probability of Zika-induced microcephaly occurring doesn't appear to be that high, but when it does, the damage is horrible."

Researchers around the world have been feverishly working to better understand the disease - which can be transmitted both by mosquito bite and through a sexual partner - and also to develop medical treatments. Tang, Ming and Song first met in graduate school 20 years ago and got in contact in January because Tang, a virologist, had access to the virus and Ming and Song, neurologists, had cortical stem cells that scientists needed to test.

The group worked at a breakneck pace with researchers from Ming and Song's lab, traveling back and forth between Baltimore and Tang's lab in Tallahassee where they had infected the cells with the virus.

In early March, the group was the first team to show that Zika indeed caused cellular phenotypes consistent with microcephaly, a severe birth defect where babies are born with a much smaller head and brain than normal. They immediately delved into follow-up work and teamed with NIH's Zheng, an expert on drug compounds, to find potential treatments for the disease.

Researchers screened 6,000 compounds that were either already approved by the FDA or were in the process of a clinical trial because they could be made more quickly available to people infected by Zika.

"It takes years if not decades to develop a new drug," Song said. "In this sort of global health emergency, we don't have time. So instead of using new drugs, we chose to screen existing drugs. In this way, we hope to create a therapy much more quickly."

All of the researchers are continuing the work on the compounds and hope to begin testing the drugs on animals infected with Zika in the near future.

The research was supported by the National Institutes of Health, Florida State University, Emory University and the Maryland Stem Cell Research Fund.

Other institutions contributing to the research are the Zhejiang University School of Medicine in China, Emory University and the Icahn School of Medicine. Emily Lee, a Florida State University graduate student working with Tang, shared the first authorship position with Assistant Professor of Biology at Emory Zhexing Wen and NIH scientist Miao Xu.

http://bit.ly/2c1k01z

Scientists report on safe, non-addictive opioid analgesic in animal model

Since the isolation of morphine from opium in the 19th century, scientists have hoped to find a potent opioid analgesic that isn't addictive and doesn't cause respiratory arrest with increased doses.

WINSTON-SALEM, N.C. - Now scientists at Wake Forest Baptist Medical Center report that in an animal model a novel pain-killing compound, BU08028, is not addictive and does not have adverse respiratory side effects like other opioids. The research findings are published in the Aug. 29 online edition of the Proceedings of the National Academy of Sciences.

"Based on our research, this compound has almost zero abuse potential and provides safe and effective pain relief," said Mei-Chuan Ko, Ph.D., professor of physiology and pharmacology at Wake Forest Baptist and lead author of the study. "This is a breakthrough for opioid medicinal chemistry that we hope in the future will translate into new and safer, non-addictive pain medications."

Pain, a symptom of numerous clinical disorders, afflicts millions of people worldwide. Despite the remarkable advances in the identification of novel targets as potential analgesics in the last decade, including nociceptin-orphanin FQ peptide (NOP) receptor, mu opioid peptide (MOP) receptor agonists remain the most widely used drugs for pain management even though they are addictive and have a high mortality rate caused by respiratory arrest, Ko said.

This study, which was conducted in 12 non-human primates, targeted a combination of classical (MOP) and non-classical (NOP) opioid receptors. The researchers examined behavioral, physiological and pharmacologic factors and demonstrated that BU08028 blocked the detection of pain without the side effects of respiratory depression, itching or adverse cardiovascular events.

In addition, the study showed pain relief lasted up to 30 hours and repeated administration did not cause physical dependence.

"To our knowledge, this is the only opioid-related analgesic with such a long duration of action in non-human primates," Ko said. "We will investigate whether other NOP/Mop receptor-related compounds have similar safety and tolerability profiles like BU08028, and initiate investigational new drug-enabling studies for one of the compounds for FDA's approval."

This study was supported by the National Institutes of Health, National Institute on Drug Abuse, grants DA023281, DA032568 and DA035359, and the U.S. Department of Defense contract W81XWH-13-2-0045.

Co-authors are: Huiping Ding, Ph.D., Paul W. Czoty, Ph.D., Norikazu Kiguchi, Ph.D., Devki D. Sukhtankar, Ph.D., Michael A. Nader, Ph.D., of Wake Forest Baptist; and Gerta Cami-Kobeci, Ph.D., and Stephen M. Husbands, Ph.D., of the University of Bath, United Kingdom.

http://bit.ly/2bNEpp8

Study finds shark fins & meat contain high levels of neurotoxins linked to Alzheimer's disease

UM research team says restricting shark consumption protects human health and shark populations

MIAMI--In a new study, University of Miami (UM) scientists found high concentrations of toxins linked to neurodegenerative diseases in the fins and muscles of 10 species of sharks. The research team suggests that restricting consumption of sharks can have positive health benefits for consumers and for shark conservation, since several of the sharks analyzed in the study are threatened with extinction due to overfishing.

Fins and muscle tissue samples were collected from 10 shark species found in the Atlantic and Pacific Oceans for concentrations of two toxins--mercury and β-N-methylamino-L-alanine (BMAA). "Recent studies have linked BMAA to neurodegenerative diseases such as Alzheimer's disease and amyotrophic lateral sclerosis (ALS)," said Deborah Mash, Professor of Neurology and senior author of the study.

Researchers at the UM Rosenstiel School of Marine and Atmospheric Science and UM Miller School of Medicine detected concentrations of mercury and BMAA in the fins and muscles of all shark species at levels that may pose a threat to human health. While both mercury and BMAA by themselves pose a health risk, together they may also have synergistic toxic impacts.

"Since sharks are predators, living higher up in the food web, their tissues tend to accumulate and concentrate toxins, which may not only pose a threat to shark health, but also put human consumers of shark parts at a health risk," said the study's lead author Neil Hammerschlag, a research assistant professor at the UM Rosenstiel School and UM Abess Center for Ecosystem Science and Policy.

Shark products including shark fins, cartilage and meat are widely consumed in Asia and globally in Asian communities, as a delicacy and as a source of traditional Chinese medicine. In addition, dietary supplements containing shark cartilage are consumed globally.

Recently scientists have found BMAA in shark fins and shark cartilage supplements. The neurotoxic methyl mercury has been known to bioaccumulate in sharks over their long lifespans.

About 16 percent of the world's shark species are threatened with extinction. The shark species sampled in this study range in threat status from least concern (bonnethead shark) to endangered (great hammerhead) by the International Union for Conservation of Nature (IUCN).

"Our results suggest that humans who consume shark parts may be at a risk for developing neurological diseases." said Mash.

"People should be aware and consider restricting consumption of shark parts. Limiting the consumption of shark parts will have positive health benefits for consumers and positive conservation outcomes for sharks, many of which are threatened with extinction due in part to the growing high demand for shark fin soup and, to a lesser extent, for shark meat and cartilage products." said Hammerschlag.

The study, titled "Cyanobacterial Neurotoxin BMAA and Mercury in Sharks," was published in Aug. 16 in the journal Toxins. The study's coauthors include: Neil Hammerschlag; David A. Davis, Kiyo Mondo, Matthew S. Seely, and Deborah C. Mash from the UM Miller School of Medicine's Department of Neurology; Susan J. Murch and William Broc Glover from the

University of British Columbia; and Timothy Divoll and David C. Evers from the Biodiversity Research Institute in Maine. The Herbert W. Hoover Foundation provided the funding for this study.

http://bit.ly/2bwwthb

Dogs understand both vocabulary and intonation of human speech

Dogs have the ability to distinguish vocabulary words and the intonation of human speech through brain regions similar to those that humans use, a new study reports.

Attila Andics et al. note that vocabulary learning "does not appear to be a uniquely human capacity that follows from the emergence of language, but rather a more ancient function that can be exploited to link arbitrary sound sequences to meanings." Words are the basic building blocks of human languages, but they are hardly ever found in nonhuman vocal communications.

Trained dogs are around the fMRI scanner. This material relates to a paper that appeared in the Sept. 2, 2016, issue of Science, published by AAAS. The paper, by A. Andics at Eötvös Loránd University in Budapest, Hungary, and colleagues was titled, "Neural mechanisms for lexical processing in dogs." **Enik Kubinyi**

Intonation is another way that information is conveyed through speech, where, for example, praises tend to be conveyed with higher and more varying pitch. Humans understand speech through both vocabulary and intonation. Here, Andics and colleagues explored whether dogs also depend on both mechanisms. Dogs were exposed to recordings of their trainers' voices as the trainers spoke to them using multiple combinations of vocabulary and intonation, in both praising and neutral ways. For example, trainers spoke praise words with a praising intonation, praise words with a neutral intonation, neutral words with a praising intonation, and neutral words with neutral intonation. Researchers used fMRI to analyze the dogs' brain activity as the animals listened to each combination. Their results reveal that, regardless of intonation, dogs process vocabulary, recognizing each word as distinct, and further, that they do so in a way similar to humans, using the left hemisphere of the brain. Also like humans, the researchers found that dogs process intonation separately from vocabulary, in auditory regions in the right hemisphere of the brain. Lastly, and also like humans, the team found that the dogs relied on both word meaning and intonation when processing the reward value of utterances. Thus, dogs seem to understand both human words and intonation. The authors note that it is possible that selective forces during domestication could have supported the emergence of the brain structure underlying this capability in dogs, but, such rapid evolution of speech-related hemispheric asymmetries is unlikely. Humans, they say, are only unique in their ability to invent words.

http://bit.ly/2byq0TG

Researchers identify characteristic chemical signature for chronic fatigue syndrome

Discovery, along with revealed underlying biology, could lead to faster, more accurate diagnoses and more effective, personalized therapies

Chronic fatigue syndrome (CFS) is a mysterious and maddening condition, with no cure or known cause. But researchers at the University of California San Diego School of Medicine, using a variety of techniques to identify and assess targeted metabolites in blood plasma, have identified a characteristic chemical signature for the debilitating ailment and an unexpected underlying biology: It is similar to the state of dauer, and other hypometabolic syndromes like caloric restriction, diapause and hibernation.

Dauer is the German word for persistence or long-lived. It is a type of stasis in the development in some invertebrates that is prompted by

harsh environmental conditions. The findings are published online in the August 29 issue of PNAS.

"CFS is a very challenging disease," said first author Robert K. Naviaux, MD, PhD, professor of medicine, pediatrics and pathology and director of the Mitochondrial and Metabolic Disease Center at UC San Diego School of Medicine. "It affects multiple systems of the body. Symptoms vary and are common to many other diseases. There is no diagnostic laboratory test. Patients may spend tens of thousands of dollars and years trying to get a correct diagnosis."

As many as 2.5 million Americans are believed to have CFS. It most often afflicts women in their 30s to 50s, though both genders and all ages can be affected. The primary symptom is severe fatigue lasting at least six months, with corollary symptoms ranging from muscle pain and headaches to sleep and memory problems.

Naviaux and colleagues studied 84 subjects: 45 men and women who met the diagnostic criteria for CFS and 39 matched controls. The researchers targeted 612 metabolites (substances produced by the processes of metabolism) from 63 biochemical pathways in blood plasma. They found that individuals with CFS showed abnormalities in 20 metabolic pathways. Eighty percent of the diagnostic metabolites measured were decreased, consistent with hypometabolic syndrome or reduced metabolism. The diagnostic accuracy rate exceeded 90 percent.

"Despite the heterogeneity of CFS, the diversity of factors that lead to this condition, our findings show that the cellular metabolic response is the same in patients," said Naviaux. "And interestingly, it's chemically similar to the dauer state you see in some organisms, which kicks in when environmental stresses trigger a slow-down in metabolism to permit survival under conditions that might otherwise cause cell death. In CFS, this slow-down comes at the cost of long-term pain and disability."

Naviaux said the findings show that CFS possesses an objectively identifiable chemical signature in both men and women and that targeted metabolomics, which provide direct small molecule information, can provide actionable treatment information. Only 25 percent of the metabolite disturbances found in each person were needed for the diagnosis of CFS. Roughly 75 percent of abnormalities were unique to each individual, which Naviaux said is useful in guiding personalized treatment.

"This work opens a fresh path to both understanding the biology of CFS and, more importantly to patients, a robust, rational way to develop new therapeutics for a disease sorely in need of them."

The study authors noted additional research using larger groups of participants from diverse geographical areas is needed to validate both the universality and specificity of the findings.

Co-authors include: Jane C. Naviaux, Kefeng Li, A. Taylor Bright, William A. Alaynick, and Lin Wang, all at UC San Diego; and Asha Baxter, Neil Nathan, Wayne Anderson, and Eric Gordon, Gordon Medical Associates.

http://bit.ly/2bAQQ7k

Five-year study reveals patients operated on at night twice as likely to die as patients who have daytime operations

Patients who have surgery during the night are twice as likely to die as patients operated on during regular working hours

New research presented at this year's World Congress of Anaesthesiologists (WCA) in Hong Kong (28 Aug - 2 Sept) shows that patients who have surgery during the night are twice as likely to die as patients operated on during regular working hours. Patients operated on later in the working day or in the early evening also have a higher mortality risk, concludes the study by Dr Michael Tessler, Associate Professor of Anesthesiology, and Dr Ning Nan Wang, Chief Resident, Department of Anesthesia at McGill University Health Centre, Montreal, Canada, and colleagues.

Postoperative mortality risk factors have been previously extensively studied. Previously identified risk factors include the patient age (1,2); the American Society of Anaesthesiologists (ASA) overall risk score

(2) and emergency status (1,2). Research studies analysing the time of surgery and postoperative mortality have had ambiguous results. The aim of this study was to investigate relationship between postoperative mortality and the time of the day of surgery at a Canadian hospital - the Jewish General Hospital in Montreal, Canada.

After obtaining institutional ethics review board approval, a retrospective review of 30 day postoperative in hospital mortality was carried out at the hospital, which is also a teaching hospital. The study evaluated all surgical procedures for the past 5 years, starting from April 1, 2010 to March 31, 2015. A database was constructed collecting variables about surgical interventions. All elective and emergent surgical cases were included except ophthalmic and local anaesthesia cases (since the vast majority of ophthalmic cases are performed under local rather than general anesthesia, and not in the regular operating theatre).

The working day was divided into three time blocks (Daytime 07:30-15:29, Evening 15:30-23:29 and night time 23:30-07:29). The start time of the anaesthetic recorded by the circulating nurse was used to determine in which time block the operation began.

There were 41,716 elective and emergency surgeries performed on 33,942 patients in 40,044 hospitalizations. Of these, 10,480 were emergency procedures; there were 3,445; 4,951; and 2,084 emergency procedures with anaesthesia starting between day, evening and night respectively. There were 226, 97 and 29 deaths of all cases during day, evening and night surgery (79, 95, 29 mortalities for emergency surgery in the same time periods) respectively.

The researchers found that after adjustment for age and ASA scores, the patients operated in the night were 2.17 times more likely to die than those operating on during regular daytime working hours, and patients operated on in the late day were 1.43 times more likely to die than those operated on during regular daytime working hours.

The researchers say: "This study demonstrates that late day and night emergency surgery are associated with higher mortality when factoring in ASA score and patient age. Postoperative 30-day in-hospital mortality rate should include start time of anaesthesia, along with other known variables, as a risk factor."

They say that theoretical possible causes include, but are not limited to, provider fatigue during anaesthesia and surgery, overnight hospital staffing issues, delays in treatment (for example how many operating rooms are available), or the patient being too sick to be postponed prior to treatment. The authors say: "Analysis of each of these possibilities is important to understand the reasons for this increased mortality and to direct any remedial action in an effort to reduce postoperative mortality."

http://cnet.co/2c4u8W7

Lightning storm kills over 300 deer in Norway

It's the biggest group of deer that has ever been killed by lightning in a single storm.

Lightning is wild and beautiful, but also very dangerous -- especially to reindeer, it seems. On August 26, a lightning storm swept the mountain plateau of Hardangervidda in Norway, laying waste to an entire herd of migrating reindeer, some 323 animals.

The kill count included 70 calves, according to the Norwegian Environment Agency's Nature Inspectorate. Five animals were still alive and had to be euthanised. Although it is not certain how the reindeer died, the Inspectorate believes that an unusually high electrical discharge interacted with the storm's highly conductive torrential rain and electrocuted them. Because reindeer huddle together during storms, the animals were found contained in an area just 50 to 80 metres (164 to 262 feet).

"We are not familiar with any previous happening on such a scale," the Inspectorate's Kjartan Knutsen told The New York Times. "Individual animals do from time to time get killed by lightning, and there are incidents where sheep have been killed in groups of 10 or even 20, but we have never seen anything like this."

http://bit.ly/2bALNbi

Researchers discover machines can learn by simply observing

Researchers working with swarm robots make major breakthrough

It is now possible for machines to learn how natural or artificial systems work by simply observing them, without being told what to look for, according to researchers at the University of Sheffield.

This could mean advances in the world of technology with machines able to predict, among other things, human behaviour.

The discovery takes inspiration from the work of pioneering computer scientist Alan Turing, who proposed a test, which a machine could pass if it behaved indistinguishably from a human. In this test, an interrogator exchanges messages with two players in a different room: one human, the other a machine.

The interrogator has to find out which of the two players is human. If they consistently fail to do so - meaning that they are no more successful than if they had chosen one player at random - the machine has passed the test, and is considered to have human-level intelligence.

Dr Roderich Gross from the Department of Automatic Control and Systems Engineering at the University of Sheffield, said: "Our study uses the Turing test to reveal how a given system - not necessarily a human - works. In our case, we put a swarm of robots under surveillance and wanted to find out which rules caused their movements. To do so, we put a second swarm - made of learning robots - under surveillance too. The movements of all the robots were recorded, and the motion data shown to interrogators."

He added: "Unlike in the original Turing test, however, our interrogators are not human but rather computer programs that learn by themselves. Their task is to distinguish between robots from either swarm. They are rewarded for correctly categorising the motion data from the original swarm as genuine, and those from the other swarm as counterfeit. The learning robots that succeed in fooling an interrogator - making it believe their motion data were genuine - receive a reward."

Dr Gross explained the advantage of the approach, called 'Turing Learning', is that humans no longer need to tell machines what to look for.

"Imagine you want a robot to paint like Picasso. Conventional machine learning algorithms would rate the robot's paintings for how closely they resembled a Picasso. But someone would have to tell the algorithms what is considered similar to a Picasso to begin with. Turing Learning does not require such prior knowledge. It would simply reward the robot if it painted something that was considered genuine by the interrogators. Turing Learning would simultaneously learn how to interrogate and how to paint."

Dr Gross said he believed Turing Learning could lead to advances in science and technology.

"Scientists could use it to discover the rules governing natural or artificial systems, especially where behaviour cannot be easily characterised using similarity metrics," he said.

"Computer games, for example, could gain in realism as virtual players could observe and assume characteristic traits of their human counterparts. They would not simply copy the observed behaviour, but rather reveal what makes human players distinctive from the rest."

The discovery could also be used to create algorithms that detect abnormalities in behaviour. This could prove useful for the health monitoring of livestock and for the preventive maintenance of machines, cars and airplanes.

Turing Learning could also be used in security applications, such as for lie detection or online identity verification.

So far, Dr Gross and his team have tested Turing Learning in robot swarms but the next step is to reveal the workings of some animal collectives such as schools of fish or colonies of bees. This could lead to a better understanding of what factors influence the behaviour of these animals, and eventually inform policy for their protection.

http://bit.ly/2bUBLjD

Doctors usually think bigger hospitals offer better surgery. Turns out, we're wrong.

We usually think large, academic hospitals are best. That's not always right.

Updated by Andrew M. Ibrahim on August 30, 2016, 7:50 a.m. ET

During surgical training I got a call from my mother with an unusually focused question. She had just returned from the doctor's office where she was told she needed to have her gallbladder removed. Her question to me was simple enough-"Where should I have my surgery?"

Like nearly every health care provider, I'm quite used to having family members ask for medical advice. Usually it is in other fields I do not practice, and I often guide them back to the doctor they're already seeing.

But this time was different. Not only was my mom asking about a procedure I have actually performed, she was also asking an important question about variation in hospital quality that is the focus of my research work. Even if I weren't her son, I was probably a good person to ask and should have an informed answer for her. Like every son, I wanted my mother to have the best operation possible. I told her to travel across town to the large academic center. She resisted.

My mother preferred to stay closer to home at a small community hospital. She argued that she was a fairly healthy person for her age and the operation was less complex than other procedures. "When I need a big heart operation, then I'll come across town," she told me.

It turns out my mom's hunch was right: My colleagues and I have found through research that big, academic medical centers don't outperform local hospitals when it comes to common procedures. This upends some of health care's most conventional wisdom — that the places with the highest volume of care provide the best quality — and suggests a different mindset for patients shopping for care.

For millions of Americans who undergo surgery every year, choosing a location of care can be challenging. The publicly reported quality measures of hospitals are difficult to understand. Even if you ask a group of surgeons for surgery recommendations, they often disagree. When I sought out advice from colleagues on where my mother should have her operation, everyone had an opinion.

Not surprisingly, they were all different and backed by little research evidence. Most did rely on the time honored adage in medicine, "the more often you do it, the better you are" that informed the advice that my mother should travel to a large high-volume academic center.

But a closer look at the research of hospital volume and outcomes reveals gaps in the logic. The most often cited papers exploring this question studied large complex operations like open heart surgery or removal of cancers. For more common procedures, like removing a gallbladder, we have little information to tell us whether our local hospital is safe, or if instead we should trek across town to a larger center. We all have just been assuming that even for these common operations, "bigger hospitals are better."

Around the time of my mother's operation, I kept reading research about variation in hospital quality for surgery, including this piece in the *New England Journal of Medicine*. The authors found that your chance of dying in the hospital after inpatient surgery could be twice as high, just based on what hospital you went to!

Of course my gut sank, and I couldn't help but wonder if I let my mother go to the wrong hospital. But when I read through their methods more closely, I still didn't have an answer. Like other evidence evaluating surgical quality, this study also focused on "high risk" operations that did not include common procedures like the one my mother needed. Thus, the question of where to go for common operations was left unanswered.

Recently in the *Journal of the American Medical Association* I reported new evidence that helps answer where you should go to obtain care for common surgical procedures.

Studying 1.6 million operations reveals something crucial about how to have safe surgery

With my colleagues at the University of Michigan, we studied 1.6 million Americans who underwent common surgical procedures including removal of the appendix, removal of the gallbladder, hernia repair, and removal of all or part of the colon. We compared those who had their operation at small, often rural hospitals with less than 25 beds (designated "critical access hospitals") with the larger, usually urban centers.

Our findings were unexpected. For these common operations, the small critical access hospitals had no difference in rates of mortality and lower rates of complications (e.g., heart attacks, pneumonias) after surgery compared with the larger hospitals. Of note, the small critical access hospitals generally operated on less-sick patients. Even when we accounted for that in our models, the findings were the same.

The findings of same or better care at smaller hospitals are surprising to most surgeons I've spoken with. After all, nearly all of us trained at large academic centers where we performed rare, complex operations and usually only saw patients from smaller hospitals when something had gone wrong. Unless we spent time in the small rural hospitals, it was easy to forget to that many of the operations performed there are less complex and happen with few complications.

Small hospitals can deliver better care at a lower cost

We also had one other unexpected finding — these small critical access hospitals cost Medicare less. For example, for a gallbladder removal at a large academic center Medicare paid approximately $13,000, whereas the critical access hospitals were paid only about $11,000 for the same operation.

This is even taking into account regional wage indexes, and that these small rural hospitals often get federal subsidies to help keep them open. For the 59 million Americans living in rural communities, this may be the most important finding affecting their future access to care. As more than 600 small rural hospitals are in danger of closing due to financial hardship, legislators are debating the value these small centers provide. By providing safe local care at lower cost, these small rural hospitals may in fact be the type of providers that legislators want to support and help keep open.

What my mom's surgery taught me, a doctor, about health care safety

My mother ended up having her operation at her local hospital and it went just fine. She got a safe operation without the trouble of traveling. It turns out, as this evidence suggests, that her experience mirrors that of most who similarly opt to stay local. For the patients that do have a complication and need a return trip the hospital, additional evidence suggests they seem to do better when they go back to the same place they had their original operation. For many patients, that could be facilitated if their first operation were closer to home.

Recently a colleague at work asked for advice about where his family member should have surgery. Many of us repeated the usual advice — "bigger hospitals are always better" — and suggested a trip across town. As our health care system attempts to deliver more patient-centered care, we'll need more research to understand which operations can be safely provided locally. For now, at least, the evidence suggests what my mother seemed to know all along — for common surgical procedures in less-complex patients, the answer is closer to home than you think.

http://bit.ly/2bRbX8K

Caffeine and its analogues revert memory deficits by normalizing stress responses in the brain

A study published in the journal Scientific Reports from Nature publishing group, describes the mechanism by which caffeine counteracts age-related cognitive deficits in animals.

The study coordinated by Portuguese researchers from Instituto de Medicina Molecular (iMM Lisboa) and collaborators from Inserm in Lille, France, along with teams from Germany and United States, showed that the abnormalexpression of a particular receptor - the

adenosine A2A, target for caffeine - in the brain of rats induces an aging-like profile namely memory impairments linked to the loss of stress controlling mechanisms.

"This is part of a larger study initiated 4 years ago in which we identified the role of this receptor in stress, but we did not know whether its activation would be sufficient to trigger all the changes. We now found that by altering the amount of this receptor alone in neurons from hippocampus and cortex - memory related areas - is sufficient to induce a profile that we designate as 'early-aging' combining the memory loss and an increase in stress hormones in plasma (cortisol)" - explains Luisa Lopes, Group Leader at iMM Lisboa and the coordinator of the study.

When the same animals were treated with a caffeine analogue, which blocks the action of adenosine A2A receptors, both memory and stress related deficits were normalized.

David Blum, from Inserm research director, adds: "In elderly people, we know there is an increase of stress hormones that have an impact on memory. Our work supports the view that the procognitive effects of A2AR antagonists, namely caffeine, observed in Alzheimer's and age-related cognitive impairments may rely on this ability to counteract the loss of stress controlling mechanisms that occurs upon aging"

This is important not only to understand the fundamental changes that occur upon aging, but it also identifies the dysfunctions of the adenosine A2A receptor as a key player in triggering these changes. And a very appealing therapeutic target" - concludes Luisa Lopes.

http://bit.ly/2bzYu7v

Tasmanian devils evolve to resist deadly cancer

Study could direct future research on cancer remission, recurrence

PULLMAN, Wash. - Tasmanian devils are evolving in response to a highly lethal and contagious form of cancer, a Washington State University researcher has found.

Andrew Storfer, WSU professor of biology, and an international team of scientists discovered that two regions in the genomes of Australia's iconic marsupials are changing in response to the rapid spread of devil facial tumor disease (DFTD), a nearly 100 percent fatal and transmissible cancer first detected in 1996.

The work, published in Nature Communications, suggests some Tasmanian devil populations are evolving genetic resistance to DFTD that could help the species avoid extinction. Additionally, the genomic data will support future medical research exploring how animals evolve rapidly in response to cancer and other pathogens.

"Our study suggests hope for the survival of the Tasmanian devil in the face of this devastating disease," Storfer said. "Ultimately, it may also help direct future research addressing important questions about the evolution of cancer transmissibility and what causes remission and reoccurrence in cancer and other diseases."

Disease kills 80 percent

Tasmanian devils are the largest carnivorous marsupials in the world and an integral part of Australia's natural heritage. Devils display significant aggression toward one another, which often involves biting on the face. This sometimes transmits DFTD, one of only three known forms of transmissible cancer and by far the most deadly.

Twenty years since its discovery, DFTD has wiped out an estimated 80 percent of devils in Tasmania, the only place in the world where the animals live.

By comparison, canine transmissible venereal tumor, a sexually transmitted form of cancer that only affects dogs, has been around for at least 11,000 years and is generally not fatal to domesticated animals.

Collections offer unique research opportunity

Despite models that predicted extinction, populations of Tasmanian devils at long-diseased sites persist. Storfer, an evolutionary geneticist who has studied DFTD for nearly a decade, teamed up with colleagues in the United States, Great Britain and Australia to investigate whether there was a genetic component to some of the devils' survival.

"If a disease comes in and knocks out 90 percent of the individuals, you might predict the 10 percent who survive are somehow genetically different," said study co-author Paul Hohenlohe, assistant professor of biology at the University of Idaho. "What we were looking for were the parts of the genome that show that difference."

The researchers mined a vast trove of devil DNA collected and stored before and after the outbreak of DFTD by wildlife ecologist and associate professor Menna Jones, study co-author, and her research team at the University of Tasmania.

Hopeful of breeding resistant devils

The frequency of genes in specific regions of the old DNA were compared to the frequency of genes in corresponding regions of DNA collected following DFTD emergence at three sites on Tasmania.

Storfer and colleagues identified two small genomic regions in the DNA samples from all three sites that exhibited significant changes in response to the strong selection imposed by the disease. Five of seven genes in the two regions were related to cancer or immune function in other mammals, suggesting that Tasmanian devils are indeed evolving resistance to DFTD.

The researchers are in the process of determining the specific functionality of the genomic regions identified in the study. They are hopeful that disease free devils with the apparently DFTD resistant DNA can be bred to enhance the genetic diversity of an off-island captive insurance population in case devil reintroductions are needed in the future.

http://bit.ly/2bZljjr

Japanese team unveils lunar rover

A team of Japanese researchers has unveiled a prototype flight model of a lunar rover based on an almost-completed design.

They aim to put what would be Japan's first exploration vehicle on the moon next year. The private sector team, named Hakuto, is made up of about 100 people including researchers from the space industry, Tohoku University scientists and others.

The model was unveiled on Monday. The new model is about 60 centimeters long and weighs roughly 4 kilograms. It is equipped with wheels designed to travel on all types of terrain.

Hakuto is one of 16 private sector teams competing in an international contest run by the US IT giant Google and a private foundation.

The first team to operate a rover for 500 meters or more on the lunar surface and successfully transmit images and video back to earth will be awarded 20-million dollars.

http://bit.ly/2bQx9NM

Smarter brains are blood-thirsty brains

A University of Adelaide-led project has overturned the theory that the evolution of human intelligence was simply related to the size of the brain -- but rather linked more closely to the supply of blood to the brain.

The international collaboration between Australia and South Africa showed that the human brain evolved to become not only larger, but more energetically costly and blood thirsty than previously believed.

The research team calculated how blood flowing to the brain of human ancestors changed over time, using the size of two holes at the base of the skull that allow arteries to pass to the brain.

The findings, published in the Royal Society journal Open Science, allowed the researchers to track the increase in human intelligence across evolutionary time.

"Brain size has increased about 350% over human evolution, but we found that blood flow to the brain increased an amazing 600%," says project leader Professor Emeritus Roger Seymour, from the University of Adelaide.

"We believe this is possibly related to the brain's need to satisfy increasingly energetic connections between nerve cells that allowed the evolution of complex thinking and learning.

"To allow our brain to be so intelligent, it must be constantly fed oxygen and nutrients from the blood. "The more metabolically active

the brain is, the more blood it requires, so the supply arteries are larger. The holes in fossil skulls are accurate gauges of arterial size."

The study was a new collaboration between the Cardiovascular Physiology team in the School of Biological Sciences at the University of Adelaide and the Brain Function Research Group and Evolutionary Studies Institute at the University of the Witwatersrand.

These are skull casts from human evolution. Left to right: Australopithecus afarensis, Homo habilis, Homo ergaster, Homo erectus and Homo neanderthalensis. **Roger Seymour. Casts photographed in the South Australian Museum.**

Co-author Dr Edward Snelling, University of the Witwatersrand, says: "Ancient fossil skulls from Africa reveal holes where the arteries supplying the brain passed through.

The size of these holes show how blood flow increased from three million-year-old Australopithecus to modern humans.

The intensity of brain activity was, before now, believed to have been taken to the grave with our ancestors."

Honours student and co-author Vanya Bosiocic had the opportunity to travel to South Africa and work with world renowned anthropologists on the oldest hominin skull collection, including the newly-discovered Homo naledi.

"Throughout evolution, the advance in our brain function appears to be related to the longer time it takes for us to grow out of childhood. It is also connected to family cooperation in hunting, defending territory and looking after our young," Ms Bosiocic says.

"The emergence of these traits seems to nicely follow the increase in the brain's need for blood and energy."

http://bbc.in/2bRfT9q

Ex-Army chief Dannatt 'sorry' over malaria drug Lariam

A former Army chief has apologised for allowing troops to take a controversial anti-malaria drug despite personally believing it can have "catastrophic" mental health effects.

By Joanna Gosling & Sarah Hatchard Victoria Derbyshire programme

Lord Dannatt admitted to the BBC's Victoria Derbyshire programme he would not take the drug himself. He said his own son had taken the drug and had become "extremely depressed".

The Ministry of Defence said: "The vast majority of deployed personnel already receive alternatives to Lariam."

'Very withdrawn'

Lord Dannatt said his son Bertie had suffered mental health problems after taking two doses of Lariam - the brand name for mefloquine - before visiting Africa as a civilian in the late 1990s.

He was not in the armed forces at the time, but had been prescribed the drug by his father's Army doctor. "He became extremely depressed," Lord Dannatt said, "not the person that he would normally be - a very bubbly, personable sort of individual. "He got very withdrawn, and we got very worried about him. "If that had been untreated, who knows where it would have gone."

The MoD's doctors prescribed Lariam to more than 17,000 troops between April 2007 and March 2015, although it is not the main anti-malaria drug used by the armed forces.

Lord Dannatt, who was head of the Army from 2006 to 2009, said the drug's side-effects - which can include depression and suicidal thoughts - could be "pretty catastrophic". He said: "Because Bertie had that effect, whenever I've needed anti-malarial drugs, I've said, 'I'll take anything, but I'm not taking Lariam.'"

Lord Dannatt said he was "quite content to say sorry" to troops who had taken Lariam while he was head of the Army, admitting the issue had not been treated as a priority.

Asked why soldiers had continued to be prescribed Lariam during his years in charge, he said the MoD at the time "hadn't reached a settled view on whether Lariam was more beneficial or harmful".

Lord Dannatt said: "I suppose, in that period from 2003 right through to 2014 - when we were focused on Iraq and Afghanistan, which were not malarial areas, and we weren't giving a large number of people Lariam - it probably slightly slipped off our mainstream radar.

"I think we put it on the backburner."

Lariam 'turned me into an ogre'

"Andy" - not his real name - took Lariam on the Army's tour of Sierra Leone in 2000, and says he still feels its side effects. "The effects were almost immediate... I can be a nasty, violent person and I attribute it to this drug. "Anything could be misconstrued - a look, a phrase, a word, something completely innocent in someone else's eyes - but it would be enough to trigger a reaction. A reaction you knew you were doing but you couldn't stop it. "It was as if the wiring in your brain had completely gone. "Had I known what the side effects were, I would have taken my chances with malaria. It turned me into an ogre."

Andy says he also gets "depressed to the point of suicidal thoughts". He explained the only reason he has come through such periods is that he has "a little girl now, and she needs a daddy. That's the only saving grace."

The MoD says that, since 2013, its doctors have prescribed Lariam to soldiers only following individual risk assessments. Lawyers acting for ex-soldiers seeking compensation take this to mean that before then there was no systematic requirement for this to happen.

Lord Dannatt said the MoD was afraid of opening "the floodgates" to "very expensive" claims if it admitted Lariam had harmed troops, adding that "frankly, the MoD doesn't have much money".

He said: "The right response by the MoD would be to take a generous approach, as far as Lariam is concerned, and invite those who think they have lost a loved one, or indeed an individual who believes he or she is still suffering as a result of Lariam, to put their case forward and have their case examined."

Critics of the use of Lariam by the MoD have described its effects as similar to "friendly fire", a mistaken attack by a military force on its own personnel. Lord Dannatt called this a "very fair description".

The Ministry of Defence said it had "a duty to protect our personnel from malaria, and, as the last Defence Committee report concluded, in some cases, Lariam will be the most effective way of doing that."

It added: "[Lariam] continues to be recommended as safe by Public Health England and the World Health Organisation."

The drug's manufacturers, Roche, said it "will continue to work with the Ministry of Defence to ensure that they have all the relevant information to ensure Lariam is prescribed appropriately".

http://bit.ly/2bJEFYM

Passive-smoking risk confirmed among Japanese

A group of researchers says the risk of lung cancer from secondhand smoke exposure has been confirmed among Japanese for the first time.

NHK -- They've called for a ban on smoking at indoor public spaces in Japan.

The team, led by Kota Katanoda at the National Cancer Center Japan, analyzed 9 studies on the relation between passive smoking and lung cancer among Japanese non-smokers.

The researchers used a statistical method in which results of multiple studies are analyzed and integrated.

They said secondhand smoke exposure increases the risk of lung cancer by about 30 percent.

The International Agency for Research on Cancer had already confirmed the risk in 2004.

The World Health Organization says 49 countries have banned smoking in indoor public places.

http://bit.ly/2bYhvhE

The Lancet Psychiatry: Increasing number of US adults using marijuana as fewer people perceive the drug as harmful

Findings suggest the need for improved education and prevention messages

An increasing number of US adults are using marijuana, as fewer people perceive the drug as harmful, according to a survey of over 500000 US adults conducted between 2002 and 2014 published in The Lancet Psychiatry. As marijuana has become increasingly potent over the past decade, the authors say that the findings suggest the need for improved education and prevention messages regarding the risks of marijuana.

While the study did not find an increase in the overall prevalence of marijuana use disorders (marijuana abuse or dependence) among US adults, it was not able to fully assess the impact of recent changes to state-level cannabis laws on widening use, and the authors say that continued monitoring of marijuana use and disorders at national and state-level is needed.

The authors note that the study did not look at use among children or teenagers, or the link between marijuana use and other more severe psychiatric disorders.

The study analysed data from 596500 adults aged 18 or older who took part in the annual US National Survey on Drug Use and Health (NSDUH) from 2002 to 2014. Marijuana use (defined as having used marijuana in the previous year) increased from 10.4% in 2002 to 13.3% in 2014. The proportion of adults who first started using marijuana in the previous year increased from 0.7% in 2002 to 1.1% in 2014. The prevalence of daily or near daily use (defined as people who reported using marijuana on average 5 days or more per week) increased from 1.9% to 3.5% over the same period.

This increase was associated with a decrease in the proportion of people perceiving great risk of harm from smoking marijuana once or twice a week from 50.4% to 33.3%. Changes in marijuana use and perception of harm generally began in 2007. The prevalence of marijuana use disorders (abuse or dependence) among adults in the general population remained stable at about 1.5% between 2002 and 2014, and the prevalence of marijuana use disorders among users declined (14.8% to 11%).

The authors suggest this may be because the large number of people who have started using marijuana in the past year might be using the drug less frequently and have less psychopathology than people who have used marijuana for longer.

Extrapolating this to the US population, the authors estimate that the number of adults who first used marijuana increased from 823000 in 2002 to 1.4 million in 2014 and that the overall number of marijuana users increased from 21.9 to 31.9 million. They estimate that the number of daily or near daily users was 8.4 million 2014, an increase from 3.9 million in 2002.

"Although shifts in perceived risk have historically been important predictors of adolescent marijuana trends, no previous research has examined this relationship in adults. State laws related to marijuana use in the USA have changed considerably over the past 20 years with medical marijuana now legalized in 25 states and the District of Columbia. Additionally, several jurisdictions have legalized non-medical marijuana use," says study author Dr Wilson M. Compton, National Institute on Drug Abuse, National Institutes of Health, USA [1].

"Understanding patterns of marijuana use and dependence, and how these have changed over time is essential for policy makers who continue to consider whether and how to modify laws related to marijuana and for health-care practitioners who care for patients using marijuana. Perceived risk of marijuana use is associated with high frequency of use suggesting the potential value for modifying risk perceptions of marijuana use in adults through effective education and prevention messages," he adds [1].

People who used marijuana were more likely to develop dependence if they were male, younger, had low education, were not in full time employment, had depression and used tobacco or other substances.

The authors note that the NSDUH relies on a large sample size and that the questionnaire content has remained unchanged since 2002, but as with any self-reported survey, answers may be subject to recall bias. The study did not include people who were homeless, living in shelters or who were incarcerated, meaning that rates of drug use and drug use disorders could be even higher. Importantly, the study did not look at other psychiatric disorders (such as psychosis or schizophrenia) so cannot provide information on the link between more severe psychiatric disorders and marijuana use.

Writing in a linked Comment, Professor Wayne Hall, University of Queensland, Australia, says: "These changes in the prevalence of cannabis use occurred during a period when many US states legalised cannabis for medicinal use, but before four states went on to legalise recreational cannabis use (after 2014). It is probably too soon to draw conclusions about the effects of these legal changes on rates of cannabis use and cannabis related harms, but it is likely that these policy changes will increase the prevalence and frequency of cannabis use and, potentially, cannabis use disorders in the longer term. To investigate this possibility, the USA needs to continue to monitor cannabis use and disorders in large scale surveys, such as the National Survey on Drug Use and Health and the Monitoring the Future national survey of high school students. Monitoring of cannabis use will need to address one of the major limitations of these surveys for this task, namely, that they were designed to provide nationally representative samples and do not necessarily provide representative samples of individual states. US Federal funding agencies should consider funding oversampling of representative population samples within states that have and have not legalised cannabis for recreational and medical use."

The National Surveys on Drug Use and Health were supported by contracts from the Substance Abuse and Mental Health Services Administration. This study was jointly sponsored by the Substance Abuse and Mental Health Services Administration, the National Institute on Drug Abuse, and the Office of the Assistant Secretary for Planning and Evaluation of the US Department of Health and Human Services.

[1] Quote direct from the author and cannot be found in the text of the Article.

http://www.thelancet.com/journals/lanpsy/article/PIIS2215-0366(16)30208-5/abstract

http://wb.md/2c19NUa

What Can Be Learned From a Cough?

Types of Cough and the Information They Provide

Nicholas Gross, MD, PhD

Chronic cough is one of the most common symptoms in medicine and is said to occur in 12% of the adult population.[1] Cough is such an obvious and easily observed mechanism to clear the lungs' airways that it tends to be ignored or taken for granted.

In an age of high-tech investigations, we tend to overlook the facts that not all coughs are alike and that features of a cough can be distinguished with current technology. But distinct varieties of cough can provide clues to underlying diagnoses.

Chronic cough, which is particularly troublesome to both the patient and the healthcare provider, has been investigated in a recent study.[2] Cough was induced in healthy smokers; in people with chronic obstructive pulmonary disease (COPD), asthma, or chronic cough; and in a control group of healthy patients using—in random order—inhaled airway stimulants, such as capsaicin, prostaglandin E_2 (PGE_2), bradykinin, and citric acid in increasing concentrations.

The investigators recorded the number and frequency of coughs several minutes after inhalations. They also made acoustic recordings of coughs over the subsequent 24 hours.

When challenged with capsaicin, cough was significantly more frequent in patients with COPD or asthma than in healthy nonsmokers and, most of all, in patients with undiagnosed "chronic cough." However, when challenged with PGE_2, cough responses were less pronounced in patients with COPD.

The investigators concluded that patients with COPD exhibit specific patterns of cough response that differ significantly not only from

healthy control subjects, but also from patients with asthma or chronic cough. This study shows for the first time that people with different airway diseases exhibit different patterns of sensitivity to a range of irritant stimuli, a phenomenon the investigators call "neurophenotypes."

Viewpoint

A shortcoming of this study is that the mechanism or mechanisms of any cough type are not addressed. It is hoped that these findings will lead to the search for biochemical and physiological abnormalities that can be corrected, relieving the distress of a common condition.

Cough serves to clear the airways. In response to upper airway infection, cough typically continues for no longer than 3 weeks, and often does not require any intervention.[3] Cough that lasts longer than that usually warrants a call or visit to a healthcare professional. Cough lasting longer than 8 weeks can be called "chronic" and deserves a work-up.

Among the pulmonary causes of chronic cough are asthma, eosinophilic bronchitis, gastroesophageal reflux, sinusitis, COPD, and bronchitis. But despite clinical interventions, almost half of the people who suffer from chronic cough receive no diagnosis,[4] and the great majority receive no relief from any therapy.

Identifying the different types of chronic cough could lead to more precise diagnoses, but more investigation is needed. This study is a start. By showing that there are different types of cough, we are confronted with the task of identifying cough types, "neurophenotypes," and appropriate treatments.

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http://bit.ly/2c1cTHD

Why pneumococci affect primarily humans

May be explained by special variant of a sugar molecule in the human nose

A special variant of a sugar molecule in the human nose might explain why pneumococcal infections are more common in humans than in other animals, researchers from Karolinska Institutet in Sweden report in a study published in the journal Cell Host & Microbe. The discovery can help in the search for a broader vaccine able to protect against all types of pneumococci.

The bacterium S. pneumoniae or the pneumococcus exists naturally in the noses of children and adults, but is also one of the most common causes of infectious diseases in the world, with meningitis and pneumonia being amongst the most severe. Pneumococci cause more severe infections in humans than in other mammals, something that has hitherto remained a mystery.

Nasal mucus contains a special sugar molecule - sialic acid - which pneumococci use as a source of energy for its growth and survival. With the help of an enzyme this acid is released by the bacteria and taken up into the bacterial cells for conversion to energy. In this present study, the researchers show that the sialic acid found in humans make pneumococci able to both grow better and become more resistant to the immune defence than the variant commonly found in other mammals.

Using mice with a mutation that causes them to produce the human form of the sugar, the researchers found that these mice were more prone to acquire a severe pneumococcal infection than the controls.

"We found that the human variant of the sugar molecule caused the bacteria to produce more of the enzyme that releases the sugar that pneumococci need as a source of energy," says principal investigator Professor Birgitta Henriques-Normark at the Department of Microbiology, Tumour and Cell Biology. "This can enhance the growth of pneumococci in the human mucosa. Moreover, the

increased uptake of sialic acid by the bacteria triggers their production of a protein [htrA] that counters the oxidative stress that the body's immune system uses to fight the infection."

The finding gives the researchers a clearer idea of how and why pneumococci can cause such severe infections in humans. This knowledge makes scientists better placed to develop more effective vaccines able to protect against all types of pathogenic pneumococci in humans, something current vaccines cannot do.

The study was a collaboration between researchers from Karolinska Institutet and the University of California and was financed with grants from the Knut and Alice Wallenberg Foundation, the Swedish Research Council, the Swedish Foundation for Strategic Research, ALF funding and the NIH.

Publication: 'Streptococcus pneumoniae senses a human-like sialic acid profile via the response regulator CiaR', Karina Hentrich, Jonas Lofling, Anuj Pathak, Victor Nizet, Ajit Varki, Birgitta Henriques-Normark, Cell Host & Microbe, online 1 September 2016.

http://bit.ly/2chUVys

Dengue vaccine could increase or worsen dengue in some settings

Researchers say health officials must be careful about where vaccine is used

The only approved vaccine for dengue may actually increase the incidence of dengue infections requiring hospitalization rather than preventing the disease if health officials aren't careful about where they vaccinate, new public health research published Sept. 2 in Science suggests.

Dengue typically causes a mild first infection but a far worse one if someone is infected with the virus a second time. There are four types of dengue virus, and it is thought that the body's response to the first infection leads to more severe disease upon a second infection. This has long posed challenges to scientists developing a vaccine, who worried that any candidate that failed to protect fully could raise the risk of making people sick rather than keeping them well.

In their new study, researchers from the Johns Hopkins Bloomberg School of Public Health, Imperial College London and the University of Florida re-analyzed data from vaccine trials conducted in 10 countries with more than 30,000 participants as well as recently published data on the long-term follow-up of these participants. Using that data, they developed mathematical models to understand how a vaccine rollout would affect people in countries where transmission of the disease is high, moderate or low. They found that while the vaccine can reduce illness and hospitalization by 20 to 30 percent in places where there is high transmission of dengue, it may actually significantly increase illness and hospitalization if used in locations where there is lower transmission of the virus.

The vaccine, manufactured by Sanofi-Pasteur, has been licensed in six countries so far, and multiple countries are currently considering how to use this vaccine.

"In vaccines you hope for more than 30 percent success, but it's the only vaccine available right now to slow dengue," says Isabel Rodriguez-Barraquer, MD, PhD, MHS, a research associate at the Bloomberg School and one of the study's lead authors. "If this vaccine is used correctly, many people could be spared illness and hospitalization from dengue. But we should make sure we only use it in places where our data suggest it will do more good than harm."

The new research suggests that the vaccine acts very much like a natural infection but without making recipients sick. In those who have previously been infected with dengue, the vaccine acts like a silent second infection, stimulating the immune system without the more severe symptoms that may accompany a natural second infection. In those who have not yet been infected with dengue, the vaccine causes the immune system to recognize that a first dengue infection has occurred and then when exposed to dengue in a natural setting, the body reacts as if it is getting a second infection that may be more severe.

The manufacturers of the three-dose vaccine have acknowledged their vaccine does not work well in people who haven't previously had a naturally occurring dengue infection before vaccination, the researchers say. The vaccine is not indicated for use in children under

the age of nine because they are least likely to have been exposed to dengue.

Partly based on these findings, the World Health Organization is recommending that this vaccine be used only in areas where there is a known high burden of disease.

"We should be careful in considering where and how to use this vaccine as there is still uncertainty about its impact," says Derek A.T. Cummings, PhD, a professor of biology at the University of Florida and an adjunct professor at the Bloomberg School and another of the study's authors. The authors hope that their analysis can help inform policymakers in evaluating this and other candidate dengue vaccines.

"Having a vaccine is a significant step forward for dengue control," Rodríguez-Barraquer says. "However, this vaccine is a prime example of having to seriously weigh the risks and benefits."

Dengue infects nearly 400 million people across more than 120 countries each year. Most survive with few or no symptoms, but more than two million annually develop what can be a dangerous dengue hemorrhagic fever, which kills more than 25,000 people each year. Dengue can cause a high fever, severe headaches, severe pain behind the eyes, rash and joint, muscle or bone pain. Dengue hemorrhagic fever occurs when blood leaks from blood vessels into other parts of the body, which can lead to failure of the circulatory system, shock and possibly death, without prompt treatment.

One thing that could help make decisions easier, the researchers say, would be a blood test that could identify those that have been infected in the past. Those who had been would get the vaccine; those who had not been would not be vaccinated.

"Benefits and risks of the Sanofi-Pasteur dengue vaccine: Modeling optimal deployment" was written by Neil M. Ferguson, Isabel Rodriguez-Barraquer, Ilaria Dorigatti, Luis Mier-y-Teran-Romero, Daniel J. Laydon and Derek A.T. Cummings.

The work was funded by the UK Medical Research Council, the UK National Institute of Health Research under the Health Protection Research Unit initiative, National Institute of Allergy and Infectious Diseases (R01 AI114703) and National Institute of General Medical Sciences (U54 GM088491) under the MIDAS initiative, and the Bill and Melinda Gates Foundation.

http://bit.ly/2cnXK4q

The Secret to a Breakthrough in Fighting Norovirus? Human Bile

Adding bile makes norovirus models even more realistic

When virologist Mary Estes first started studying norovirus—the bug best known for causing vomiting and diarrhea on cruise ships—she had a basic problem.

She didn't have enough virus to study. So she got more the only way scientists knew how at the time: Take stool samples from a norovirus patient, infect (brave) volunteers, and wait for them to poop out norovirus particles by the millions. That's how she first unraveled norovirus's genome.

She spent the next two years looking for a better way to grow norovirus. So did other labs. Norovirus is surprisingly finicky: It can grow like crazy in your gut, but it just will not grow in a petri dish.

So Estes, now at Baylor, recreated the human gut in a petri dish—growing stem cells that turn into little balls of gut tissue.

By adding bile, aka digestive juices, to make the model even more realistic, her team turned those mini guts into norovirus factories. This is a major breakthrough for a virus that sickens 20 million Americans a year, yet still remains fundamentally mysterious.

Without a robust way of growing norovirus outside of human volunteers, scientists can't easily check whether the virus in a sample is alive or dead.

They don't know, for example, if hand sanitizers actually kill norovirus. They don't know if the viruses shed by people who've gotten over their symptoms are still contagious or not. They can't easily develop vaccines.

Now, thanks to Estes' work, they finally can. "It's a champagne popping moment," says Jan Vinjé, a virologist at the Centers for Disease Control and Prevention, who has spent two decades studying the virus. (Vinjé has collaborated with Estes in the past but is not an author on the recent Science paper.)

Vinjé's lab at the CDC, along with two outside labs other than Estes, have been able to replicate the mini gut bile work already.

That's huge: Other methods of cultivating norovirus have grabbed headlines before, only to fade away when outside labs couldn't get it to work. Nearly a decade ago, the field got all excited about a 3D intestinal tissue model that could harbor noroviruses. Vinjé's lab could never replicate it.

"I had a post doc who worked on that for two years, who still gets frustrated if I talk about it," says Vinjé.

The success of the mini gut and norovirus could steer the whole field of infectious diseases in a new direction.

Traditionally, scientists have grown viruses in human cancer cells, which have the unusual ability to keep dividing and dividing. This is great if you want to grow a lot of cells, but not always so good if you want to study how a virus infects a healthy cell.

"The results we obtain from cell lines just don't reflect the reality of what's on the street," says Timothy Straub, a microbiologist at Pacific Northwest National Laboratory, who is growing healthy lung tissue to study inhaled pathogens.

Straub actually came up with the original 3D intestinal tissue model that so vexed Vinjé post doc. That model used cancer cells, and he now readily admits it doesn't work. He's convinced using healthy cells is the way of the future.

Estes, for her part, first came up with the idea of growing mini guts for norovirus when she was reading up on a Dutch group's work in stem cells, which doesn't seem like it should have much to do with norovirus. But when she saw that group could coax stem cells into mini guts, she made the leap to the problem she's been trying to solve for decades.

"The most important thing I've learned in my career is to read very broadly," she says. And now scientists have a much better way to grow norovirus. No pooping humans needed.

http://n.pr/2bLwCNT

An Even Deadlier Opioid, Carfentanil, Is Hitting The Streets

First responders have found that standard doses of naloxone aren't always enough to counteract the powerful sedating effects of carfentanil.

Jennifer Ludden

Download Audio

A powerful drug that's normally used to tranquilize elephants is being blamed for a record spike in drug overdoses in the Midwest. Officials in Ohio have declared a public health emergency, and the U.S. Drug Enforcement Administration says communities everywhere should be on alert for carfentanil.

The synthetic opioid is 100 times more potent than fentanyl, the prescription painkiller that led to the death earlier this year of the pop star Prince. Fentanyl itself can be up to 50 times more deadly than heroin.

In the past few years, traffickers in illegal drugs increasingly have substituted fentanyl for heroin and other opioids. Now carfentanil is being sold on American streets, either mixed with heroin or pressed into pills that look like prescription drugs. Many users don't realize that they're buying carfentanil. And that has deadly consequences.

"Instead of having four or five overdoses in a day, you're having these 20, 30, 40, maybe even 50 overdoses in a day," says Tom Synan, who directs the Hamilton County Heroin Coalition Task Force in Southwest Ohio. He's also the police chief in Newtown, Ohio.

Synan says carfentanil turned up in Cincinnati in July. At times, the number of overdoses has overwhelmed first responders. "Their efforts are truly heroic, to be going from call to call to call," he says. "One district alone had seen 14 in one shift, so they were nonstop."

First responders and emergency room workers are being told to wear protective gloves and masks. That's because carfentanil is so potent, it can be dangerous to someone who simply touches or inhales it.

This was devastatingly clear back in 2002, after a hostage rescue operation in Moscow that went wrong. To overpower Chechen terrorists who'd seized control of a theater, Russian Special Forces sprayed a chemical aerosol into the building. More than 100 hostages were overcome and died. Laboratory tests by British investigators later revealed that the aerosol included carfentanil.

In Ohio, Hamilton County Health Commissioner Tim Ingram says it can take hours for the body to metabolize carfentanil, far longer than for other opioids. That means a longer-lasting high.

But it also means that when someone overdoses, it's more difficult to revive them — and save their life — with naloxone, the emergency medication used to block the effects of opioids.

"We've been getting lots of reports that they're using two or three doses to get people to come back," says Ingram. He's trying to distribute a more concentrated version of naloxone.

There is no approved human use for carfentanil. It's even highly restricted for veterinarians, who can use it lawfully to sedate large animals. The Drug Enforcement Administration says much of the carfentanil being sold on the streets is illicitly imported from China.

DEA spokesman Russ Baer says some of the illicit carfentanil is brought in by Mexican drug traffickers, then sold at huge profit since it only takes a granule or so to induce a high. He says carfentanil can also be bought online.

"You can go on the Internet and anybody can establish an anonymous account, and you can order carfentanil directly from China," he says.

Ingram foresees a turning point in illicit opioids. He wonders why anyone would go to the trouble of growing poppies in order to make heroin, when something much more powerful can be made in a lab.

"We may be seeing more and more synthetic opioids from this point forward," he says, "and we're going to have to prepare for it."

Synan thinks one shift should include tougher penalties. Generally, he says, selling drugs on the street is considered a nonviolent crime. But that may not make sense if the drug includes carfentanil.

"To me, that's just like pulling a gun out and shooting someone, because you know that a tiny bit can kill a person," Synan says. "To me, it's intentional. It's murder."

http://bit.ly/2c2Ka74

Hereditary diseases are the price of protection against infections

Balancing selection is responsible for helping us fend off pathogens, but also for the occurrence of mutations in our genome that predispose us to hereditary diseases

Almost half our genes can be the starting point for diseases. Scientists have identified 11,000 genes that occur in the human genome in variants that can cause disease. Scientists from the Max Planck Institute for Evolutionary Biology in Plön and the Harvard Medical School have studied why such high-risk genes persist in the human genome instead of being eliminated by selection. Their analyses suggest that the continuous adaptation to new pathogens in the course of evolution has increased the diversity of our immune genes but also comes at a price. According to the researchers, such diversity also extends to neighbouring DNA segments, where it results in the persistence of harmful gene variants.

Diversity in the genome is a good thing: it has allowed us humans to adjust to changing environmental conditions during the course of evolution. Such genetic variety generates diverse combinations with each new generation and can bring with it survival advantages. Besides the many variants that have no effect or even a beneficial effect on health, there are others that make their carriers susceptible to certain diseases.

These harmful gene variants represent a survival disadvantage and should therefore have been weeded out by natural section in the course of evolution. Instead, some high-risk gene variants, such as those for Alzheimer's disease or cancer, have persisted in the population for a long time without disappearing.

A group of researchers led by Tobias Lenz and Shamil Sunyaev has studied this phenomenon and found evidence that the occurrence of harmful gene variants could be the price we pay for the genetic diversity that is otherwise highly beneficial to our survival. They analyzed a group of immune system proteins that help detect foreign molecules. The genes for these proteins contain many variable sites and occur in a number of alternative forms in the population. This diversity ensures that our immune system is able to recognize a broad range of pathogens.

A special form of selection preserves this variation within the group of immune proteins: scientists describe it as balancing selection. It arises, for example, when several alternative variants of a gene confer a survival advantage, and are therefore not eliminated by selection.

Harmful mutations don't get lost

The scientists suspect that balancing selection may sometimes also lead to the conservation of harmful gene variants. They ran computer simulations of different types of selection using the example of immune system genes. During these tests they discovered that balancing selection not only increases the diversity of immune proteins but also affects neighbouring DNA segments. There, while reducing the total number of variable sites, it increases the frequency with which these variants occur in the population - even if they are harmful.

They then compared the simulation results with data from a genetic analysis of 6,500 people. And the analysis confirmed their suspicions: As in the simulation, fewer variable sites occurred in the immediate vicinity of the immune system genes; however, the remaining variants, including harmful mutations, were relatively more common in the population.

Harmful genes are therefore able to evade natural selection. "I did expect that higher resistance to pathogens might lead to an accumulation of some harmful mutations. But the extent to which such mutations persist in the population really surprised me. It would be interesting to know how many genetic diseases in humans can be traced back to contact with pathogens we have encountered in the course of our evolution," says Tobias Lenz, group leader at the Max Planck Institute in Plön and member of the newly founded Kiel Evolution Center.

In the next step, the researchers want to examine whether balancing selection at other sites in the genome are responsible for the fact that harmful gene variants occur so frequently in the population.

Original publication: Tobias L. Lenz, Victor Spirin, Daniel M. Jordan, & Shamil R. Sunyaev Excess of deleterious mutations around HLA genes reveals evolutionary cost of balancing selection.

http://bit.ly/2bK8zJ9

For first time, carbon nanotube transistors outperform silicon

Carbon nanotube transistors created that outperform state-of-the-art silicon transistors

MADISON -- For decades, scientists have tried to harness the unique properties of carbon nanotubes to create high-performance electronics that are faster or consume less power -- resulting in longer battery life, faster wireless communication and faster processing speeds for devices like smartphones and laptops.

But a number of challenges have impeded the development of high-performance transistors made of carbon nanotubes, tiny cylinders made of carbon just one atom thick. Consequently, their performance has lagged far behind semiconductors such as silicon and gallium arsenide used in computer chips and personal electronics.

Now, for the first time, University of Wisconsin-Madison materials engineers have created carbon nanotube transistors that outperform state-of-the-art silicon transistors.

Led by Michael Arnold and Padma Gopalan, UW-Madison professors of materials science and engineering, the team's carbon nanotube transistors achieved current that's 1.9 times higher than silicon transistors. The researchers reported their advance in a paper published Friday (Sept. 2) in the journal Science Advances.

"This achievement has been a dream of nanotechnology for the last 20 years," says Arnold. "Making carbon nanotube transistors that are better than silicon transistors is a big milestone. This breakthrough in carbon nanotube transistor performance is a critical advance toward exploiting carbon nanotubes in logic, high-speed communications, and other semiconductor electronics technologies."

This advance could pave the way for carbon nanotube transistors to replace silicon transistors and continue delivering the performance gains the computer industry relies on and that consumers demand. The new transistors are particularly promising for wireless communications technologies that require a lot of current flowing across a relatively small area.

As some of the best electrical conductors ever discovered, carbon nanotubes have long been recognized as a promising material for next-generation transistors.

Carbon nanotube transistors should be able to perform five times faster or use five times less energy than silicon transistors, according to extrapolations from single nanotube measurements. The nanotube's ultra-small dimension makes it possible to rapidly change a current signal traveling across it, which could lead to substantial gains in the bandwidth of wireless communications devices.

But researchers have struggled to isolate purely carbon nanotubes, which are crucial, because metallic nanotube impurities act like copper wires and disrupt their semiconducting properties -- like a short in an electronic device.

The UW-Madison team used polymers to selectively sort out the semiconducting nanotubes, achieving a solution of ultra-high-purity semiconducting carbon nanotubes.

"We've identified specific conditions in which you can get rid of nearly all metallic nanotubes, where we have less than 0.01 percent metallic nanotubes," says Arnold.

Placement and alignment of the nanotubes is also difficult to control. To make a good transistor, the nanotubes need to be aligned in just the right order, with just the right spacing, when assembled on a wafer. In 2014, the UW-Madison researchers overcame that challenge when they announced a technique, called "floating evaporative self-assembly," that gives them this control.

The nanotubes must make good electrical contacts with the metal electrodes of the transistor. Because the polymer the UW-Madison researchers use to isolate the semiconducting nanotubes also acts like an insulating layer between the nanotubes and the electrodes, the team "baked" the nanotube arrays in a vacuum oven to remove the insulating layer. The result: excellent electrical contacts to the nanotubes.

The researchers also developed a treatment that removes residues from the nanotubes after they're processed in solution.

"In our research, we've shown that we can simultaneously overcome all of these challenges of working with nanotubes, and that has allowed us to create these groundbreaking carbon nanotube transistors that surpass silicon and gallium arsenide transistors," says Arnold.

The researchers benchmarked their carbon nanotube transistor against a silicon transistor of the same size, geometry and leakage current in order to make an apples-to-apples comparison.

They are continuing to work on adapting their device to match the geometry used in silicon transistors, which get smaller with each new generation. Work is also underway to develop high-performance radio frequency amplifiers that may be able to boost a cellphone signal. While the researchers have already scaled their alignment and deposition process to 1 inch by 1 inch wafers, they're working on scaling the process up for commercial production.

Arnold says it's exciting to finally reach the point where researchers can exploit the nanotubes to attain performance gains in actual technologies.

"There has been a lot of hype about carbon nanotubes that hasn't been realized, and that has kind of soured many people's outlook," he says. "But we think the hype is deserved. It has just taken decades of work

for the materials science to catch up and allow us to effectively harness these materials."

The researchers have patented their technology through the Wisconsin Alumni Research Foundation.

Funding from the National Science Foundation, the Army Research Office and the Air Force supported their work.

Additional authors on the paper include Harold Evensen, a University of Wisconsin-Platteville engineering physics professor, Gerald Brady, a UW-Madison materials science and engineering graduate student and lead author on the study, and graduate student Austin Way and postdoctoral researcher Nathaniel Safron.

http://bit.ly/2bLDefl

Placenta in females, muscle mass in males: The dual heritage of a virus

Genes of viral origin may also be responsible for the more developed muscle mass seen in males

It is known that genes inherited from ancient retroviruses are essential to the placenta in mammals, a finding to which scientists in the Laboratoire Physiologie et Pathologie Moleacuteculaires des Retrovirus Endogenes et Infectieux (CNRS/Universite Paris-Sud) contributed. Today, the same scientists reveal a new chapter in this story: these genes of viral origin may also be responsible for the more developed muscle mass seen in males. Their findings are published on 2 September 2016 in PLOS Genetics.

Retroviruses carry proteins on their surface that are able to mediate fusion of their envelope with the membrane of a target cell. Once released inside that cell, their genetic material becomes integrated in the host's chromosomes. In the rare cases where the infected cell is involved in reproduction, the viral genes may be transmitted to progeny. Thus nearly 8% of the mammalian genome is made up of vestiges of retroviruses, or "endogenous" retroviruses. Most of them are inactive, but some remain capable of producing proteins: this is the case of syncytins, proteins that are present in all mammals and encoded by genes inherited from retroviruses "captured" by their ancestors. A little more than five years ago, and thanks to inactivation of these genes in mice, the team led by Thierry Heidmann demonstrated that syncytins contribute to formation of the placenta. Because of their ancestral ability to mediate cell-cell fusion they give rise to the syncytiotrophoblast, a tissue formed by the fusion of a large number of cells derived from the embryo, at the fetomaternal interface. Using the same mice, the team has revealed a "collateral" and unexpected effect of these proteins: they endow males with more muscle mass than females. Like the syncytiotrophoblast, muscle mass develops from fused stem cells. In the genetically-modified male mice, these fibers were 20% smaller and displayed 20% fewer nuclei than in standard males; they were then similar to those seen in females, as was their total muscle mass. It therefore appears that the inactivation of syncytins leads to a fusion deficit during muscle growth, but only in males. The scientists observed the same phenomenon in the case of muscle regeneration following a lesion: the male mice incapable of producing syncytins experienced less effective regeneration than the other males, but it was comparable to that seen in females. Furthermore, the regenerating muscle fibers produced syncytin - once again, only in males.

If this discovery were to be confirmed in other mammals, it might account for the muscle dimorphism observed between males and females, a difference that is not seen so systematically in egg laying animals. By cultivating muscle stem cells from different mammalian species (mouse, sheep, dog, human), the scientists have advanced some way along the path: they indeed showed that syncytins contributed to the formation of muscle fibers in all the species tested. It is now necessary to demonstrate whether, in these species as well, the action of syncytins is also male-specific.

Citation: Redelsperger F, Raddi N, Bacquin A, Vernochet C, Mariot V, Gache V, et al. (2016) Genetic Evidence That Captured Retroviral Envelope syncytins Contribute to Myoblast Fusion and Muscle Sexual Dimorphism in Mice. PLOS Genetics: http://dx.plos.org/10.1371/journal.pgen.1006289

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http://bit.ly/2cxMKTl

Two Pterosaur Finds Are Helping to Untangle Their Family Tree

An intact skull and a pint-sized species offer clues to how these creatures evolved

By Jason Daley

Pterosaurs come in many different shapes and sizes. These flying reptiles ruled the skies during the age of dinosaurs. But how they fit into evolutionary history has long confused scientists. Now two new species—one the brainiest and another among the smallest—are helping researchers rework the pterosaur evolutionary tree.

The tiny pterosaur from the late Cretaceous was no bigger than a cat and sported a five-foot wingspan. **Mark Witton**

The first of these fossils was discovered in the Patagonia region of Argentina and has a surprisingly pristine skull. Delicate and light weight, pterosaur skulls are usually crushed before they fossilize—researchers have only ever found a few intact specimen.

They dubbed the species Allkaruen koi, which means ancient brain in the indigenous Tehuelche language, and made a detailed CT scan. This analysis allowed the researchers to reconstruct features of the 190-million-year-old animal's brain and inner ear, according to a press release.

From these scans, the researchers hope to figure out how the pterosaur's brain evolved over time and adapted to life on the wing. "Allkaruen, from the middle lower Jurassic limit, shows an intermediate state in the brain evolution of pterosaurs and their adaptations to the aerial environment," Diego Pol, who is part of the research team, says in the release. "As a result, this research makes an important contribution to the understanding of the evolution of all of pterosaurs."

The second pterosaur recently unveiled is a tiny creature, no bigger than a cat with a wingspan of around five feet, Eva Botkin-Kowacki reports for The Christian Science Monitor.

The creature's fused vertebrae and bone structure indicate that the fossils come from mature animals not from juveniles of larger species, according to a press release.

The creature likely lived during the late Cretaceous period, 70 to 85 million years ago. But while tiny pterosaurs were common in earlier eras, they are absent from the fossil record at this late date. Many believe that by this time the giant pterosaurs dominated, with one species sporting a 32-foot wingspan. It was also thought that, in the late Cretaceous, birds filled the tiny creatures' niche.

Allkaruen koi **Gabriel Lío**

"We've got a small pterosaur when everyone said they shouldn't be there," study co-author Elizabeth Martin-Silverstone tells Traci Watson at National Geographic.

But the researchers argue the new fossil suggests otherwise. The absence of juvenile pterosaurs in the fossil record from the larger species could mean that the remains from these tiny winged creatures just didn't survive to the present day.

Martin-Silversone suggests that perhaps some of these missing fossils are currently lurking in museum and college collections, mislabeled or yet-to-be identified.

http://nyti.ms/2bKaNbl

F.D.A. Bans Sale of Many Antibacterial Soaps, Saying Risks Outweigh Benefits

F.D.A. said it will ban triclosan, among other antibacterial chemicals

By SABRINA TAVERNISE SEPT. 2, 2016

WASHINGTON — The Food and Drug Administration banned the sale of soaps containing certain antibacterial chemicals on Friday, saying industry had failed to prove they were safe to use over the long term or more effective than using ordinary soap and water.

In all the F.D.A. took action against 19 different chemicals and has given industry a year to take them out of their products. About 40 percent of soaps — including liquid hand soap and bar soap – contain the chemicals. Triclosan, mostly used in liquid soap, and triclocarban, in bar soaps, are by far the most common.

The rule applies only to consumer hand washes and soaps. Other products may still contain the chemicals. At least one toothpaste, Colgate Total, still does, but the F.D.A. says its maker proved that the benefits of using it — reducing plaque and gum disease — outweigh the risks.

The agency is also studying the safety and efficacy of hand sanitizers and wipes, and has asked companies for data on three active ingredients — alcohol (ethanol or ethyl alcohol), isopropyl alcohol and benzalkonium chloride — before issuing a final rule on them.

Public health experts applauded the rule, which came after years of mounting concerns that the antibacterial chemicals that go into everyday products are doing more harm than good. Experts have pushed the agency to regulate antimicrobial chemicals, warning that they risk scrambling hormones in children and promoting drug-resistant infections.

“It has boggled my mind why we were clinging to these compounds, and now that they are gone I feel liberated,” said Rolf Halden, a scientist at the Biodesign Institute at Arizona State University, who has been tracking the issue for years. “They had absolutely no benefit but we kept them buzzing around us everywhere. They are in breast milk, in urine, in blood, in babies just born, in dust, in water.”

The agency first proposed the rule in 2013, when it told companies that unless they could prove that chemicals like triclosan and triclocarban did more good than harm, they would have to remove the products that contained them from the market. On Friday, the agency said that it was not convinced.

The F.D.A. has given industry more time to prove that an additional three chemicals are safe and effective — benzalkonium chloride, benzethonium chloride and chloroxylenol. Products with those chemicals can stay on the market for now.

The American Cleaning Institute, a trade group, opposed the rule, saying the agency “has in its hands data that shows the safety and effectiveness of antibacterial soaps.” The group said manufacturers were continuing to work to provide even more science and research “to fill data gaps identified by the F.D.A.”

But some of the largest companies have already started removing the chemicals, in part a reaction to rising consumer concerns. Both Johnson & Johnson and Procter & Gamble announced their intention to phase out the chemicals in their products before the rule was made final, said Dr. Theresa Michele, the director of the division of nonprescription drug products at the F.D.A.’s Center for Drug Evaluation and Research.

Studies in animals have shown that triclosan and triclocarban can disrupt the normal development of the reproductive system and metabolism, and health experts warn that their effects could be the same in humans. The chemicals were originally used by surgeons to wash their hands before operations, and their use exploded in recent years as manufacturers added them to a variety of products, including mouthwash, laundry detergent, fabrics and baby pacifiers. The Centers for Disease Control and Prevention found the chemicals in the urine of three-quarters of Americans.

Dr. Halden began publishing findings on what appeared to be risks of triclocarban in 2004. He said it is an older chemical, part of the family of organochlorines, like DDT and hexachlorophene, some of which were eventually banned. Newer chemicals are much lighter on the environment, he said, but triclocarban takes a very long time to disappear. In one study in New York City, for example, his team found traces of it that dated back to the 1960s.

“It was still sitting there in Jamaica Bay near J.F.K. Airport,” he said. “This stuff makes no sense.”

http://bbc.in/2bLFJ1k

Plastic microbeads to be banned by 2017, UK government pledges

The UK government has announced plans to ban microbeads used in cosmetics and cleaning products by 2017.

The small pieces of plastic commonly found in toothpaste, exfoliating body scrubs and other household products and are thought to damage the environment. Environmentalists fear they are building up in oceans and potentially entering the food chain.

A consultation on how a ban would work will start later this year, Environment Secretary Andrea Leadsom has announced. A number of cosmetic companies have made voluntary commitments to phase out the use of microbeads by 2020.

How do you know if a product contains microbeads?

Products that contain the tiny bits of plastic won't necessarily say "microbeads" in the list of ingredients.

Instead, look for the words polyethylene, polypropylene and polymethylmethacrylate - the chemical names for plastics. Nylon may also be listed as well as the abbreviations PET, PTFE and PMMA.

There are several websites listing products that do and do not include plastic such as Beat the Microbead. It also has a free app where you can check products by scanning the barcode with your smartphone camera.

Many cosmetics brands include information on their websites. Johnson & Johnson which produces face scrubs under the brands Neutrogena and Clean & Clear has committed to phasing out microbeads by the end of 2017.

Proctor and Gamble which owns Crest toothpaste, Gillette and Olay, has also promised to stop using them by next year.

The House of Commons Environmental Audit Committee last month said the government needed to step in to protect the environment as soon as is practicable, after it was revealed a single shower can result in 100,000 plastic particles entering the ocean.

Mrs Leadsom said: "Most people would be dismayed to know the face scrub or toothpaste they use was causing irreversible damage to the environment, with billions of indigestible plastic pieces poisoning sea creatures. "Adding plastic to products like face washes and body scrubs is wholly unnecessary when harmless alternatives can be used." She said it was the "next step in tackling microplastics in our seas" following the introduction of the 5p plastic bag charge, which was introduced in England in October.

Professor Richard Thompson, a marine biologist from Plymouth University, welcomed the decision. He said: "Over 680 tonnes of mircrobeads are used in the UK alone every year. That's substantially more than all of the litter we pick up on our beaches in voluntary beach cleans each year, so it's not a trivial quantity.

"The sooner we can make progress with avoidable, unnecessary emissions, because it's not clear to me at all why we need to cleanse ourselves by rubbing our skin with millions of small, plastic particles. **What's the societal benefit there?"**

The environment committee's report suggested microplastic pollution could be more damaging to the environment than larger pieces of plastic because its size makes it more likely to be eaten by wildlife and then potentially enter the food chain.

As an example, it said a plate of six oysters can contain up to 50 particles of plastic.

More than 280 marine species have been found to ingest microplastics, but the committee said much more research was needed into plastic pollution because there was huge uncertainty about the ecological risk. It added there was "little evidence" about the potential human health impacts of microplastic pollution, but said further research was "clearly required".

'Credit to May'

Commenting ahead of the government's move, Greenpeace UK senior oceans campaigner Louise Edge said: "It's a credit to Theresa May's government that they've listened to concerns from the public, scientists and MPs, and taken a first step towards banning microbeads. "Marine life doesn't distinguish between plastic from a face wash and plastic from a washing detergent, so the ban should be extended to microplastics in any product that could be flushed down the drain.

"If Theresa May wants to show real leadership on this issue, that's the kind of ban she should back."

The US recently became the first country to announce it would ban microbead use in cosmetics, with pressure growing globally to take action. The European Commission is also currently developing proposals to ban them in cosmetics across the EU, following calls from a number of member states.

http://nyti.ms/2bKaYnk

Study Finds Increase in Temporary Paralysis Accompanied Zika Outbreaks

Only one in five infected people show symptoms of Zika

By CATHERINE SAINT LOUIS AUG. 31, 2016

In seven countries that recently experienced Zika outbreaks, there were also sharp increases in the numbers of people suffering from a form of temporary paralysis, researchers reported Wednesday.

The analysis, published online in The New England Journal of Medicine, adds to substantial evidence that Zika infections — even asymptomatic ones — may bring on a paralysis called Guillain-Barré syndrome.

The syndrome can be caused by a number of other factors, including infection with other viruses. Researchers studying the Zika epidemic in French Polynesia had estimated that roughly 1 in 4,000 people infected with the virus could develop the syndrome.

The Centers for Disease Control and Prevention has said that the Zika virus is “strongly associated” with Guillain-Barré, but has stopped short of declaring it a cause of the condition.

The new data suggest a telling pattern: Each country in the study saw unusual increases in Guillain-Barré that coincided with peaks in Zika infections, the researchers concluded.

“It’s pretty obvious that in all seven sites there is a clear relationship,” said Dr. Marcos A. Espinal, the study’s lead author and the director of communicable diseases at the Pan American Health Organization, which collected data on confirmed and suspected cases of Zika infection and on the incidence of Guillain-Barré. “Something is going on.”

In Venezuela, officials expected roughly 70 cases of Guillain-Barré from December 2015 to the end of March 2016, as mosquitoes were spreading the virus. Instead, there were 684 cases.

Similarly, during five months in which the Zika virus was circulating in Colombia, officials recorded 320 cases of Guillain-Barré when there should have been about 100. From September 2015 to March 2016, while Zika infections peaked in El Salvador, cases of Guillain-Barré doubled to 184 from 92.

The researchers included patients with both suspected and confirmed Zika infections, as reported by national health officials.

Dr. Kenneth C. Gorson, professor of neurology at Tufts University School of Medicine, who was not involved with the new analysis, called it compelling.

“This is a substantial public health burden for countries that may not have well-developed health systems in place,” he said. “They have to have enough ventilators and I.C.U. beds.” About one-third of patients with Guillain-Barré require breathing assistance, he said.

Over all, Dr. Espinal and his authors found increases in Guillain-Barré that were two to 10 times what would normally be expected. Roughly 500 million people in Latin America and the Caribbean are at risk for Zika virus infection, so even modest increases in the incidence of Guillain-Barré are worrisome.

The nations in the study included the Dominican Republic, El Salvador, Honduras, Suriname, Venezuela and Colombia, along with the state of Bahia in Brazil. (National data from Brazil was not available until February 2016.)

Collectively, they reported a total of nearly 1,500 cases of temporary paralysis. The reported incidence was 28 percent higher for men and increased with age for both sexes, in line with previous research.

Temporary paralysis is a potential neurological complication of dengue infection, too. But Dr. Espinal and his colleagues looked for a similar link to dengue and found none.

Dr. Gorson noted that the continental United States has no formal monitoring system for Guillain-Barré. As the number of Zika cases in Florida and elsewhere increases, he said, “you won’t know if a Guillain-Barré case is related to Zika infection.”

“We can do it,” he said of such surveillance. But “there’s no funding from Congress to do it.”

http://bit.ly/2bQM6xs

Lung cancer patients whose tumor has spread to the brain could be spared radiotherapy

Brain radiotherapy makes little or no difference to how length of survival and quality of life

PATIENTS with non-small cell lung cancer which has spread to the brain could be spared whole brain radiotherapy as it makes little or no difference to how long they survive and their quality of life, according to a Cancer Research UK-funded clinical trial published today (Sunday) in The Lancet*.

Around 45,500 people are diagnosed with lung cancer in the UK every year and an estimated 85 per cent of cases are non-small cell lung cancer. Up to 30 per cent of patients with non-small cell lung cancer have the disease spread to the brain.

Typically these patients are given steroids and supportive care, such as painkillers, to control their cancer symptoms, but may also be offered whole brain radiotherapy daily for one to two weeks to improve symptoms. Before this trial, doctors had little evidence to prove whether giving these patients whole brain radiotherapy benefitted them.

Because whole brain radiotherapy can cause side effects** and involves daily visits to the hospital, the QUARTZ trial*** looked at whether it improves how long patients survived for and its effect on quality of life.

The trial, led by researchers from the MRC Clinical Trials Unit at UCL, studied 538 patients from the UK and Australia. Half of the patients had whole brain radiotherapy and the other half did not, all the patients received steroids and supportive care. The trial found no clear difference in survival and quality of life between the patients who did and didn't receive whole brain radiotherapy.

The patients who had whole brain radiotherapy lived for around five days longer (9.2 weeks after entering the trial compared with 8.5 weeks for those who didn't receive radiotherapy), and reported around five more days of good quality life****. These small differences could be down to chance and suggest that whole brain radiotherapy doesn't increase survival or quality of life. This means that patients could be spared the extra radiotherapy treatment.

While research has doubled cancer survival rates, progress has not been the same across all cancer types and survival remains low for people with lung cancer. To help tackle this Cancer Research UK increased investment in this cancer in its research strategy in 2014.

Dr Paula Mulvenna, the clinical chief investigator from the Northern Centre for Cancer Care in Newcastle, said: "This trial is changing treatment for patients. Before the QUARTZ trial clinicians weren't certain that giving whole brain radiotherapy enhanced our patients'

quality of life, but did frequently offer it in good faith. These results confirm we can safely omit this treatment and concentrate on other ways of ensuring our patients and their families receive the best end of life care."

Professor Ruth Langley, from the MRC Clinical Trials Unit at UCL, said: "We're extremely grateful to the patients, carers and clinicians who took part in this challenging trial and helped us identify this important information that could improve the final days for many patients around the world."

Martin Ledwick, Cancer Research UK's head information nurse, said: "These trial results could help patients with limited time choose how they spend the end of their lives. For many people spending time at home with family and friends is their priority so knowing that they can do this rather than going backwards and forwards to hospital could be their preference."

** Mulvenna et al. Can whole brain radiotherapy be omitted from the treatment of non small cell lung cancer patients with brain metastases not amenable to stereotactic radiotherapy or surgery? Results from the UK Medical Research Council QUARTZ randomised clinical trial. The Lancet. 2016.*

*** These include hair loss, headache, tiredness, nausea, clumsiness, a dry or itchy scalp and poor concentration.*

**** More information available here: http://www.cancerresearchuk.org/about-cancer/find-a-clinical-trial/a-trial-looking-at-the-treatment-of-lung-cancer-which-has-spread-to-the-brain*

***** The trial measured quality adjusted life years (QALYs) which takes into account both length of survival and quality of life during that time. On average, those who had steroids, supportive care and whole brain radiotherapy had 46.4 days of good quality of life, and those who had steroids and supportive care 41.7 days of good quality life, a difference of 4.7 days.*

http://bit.ly/2c12n09

Asthma gene's effect on respiratory symptoms in infancy depends on breastfeeding status

Breastfeeding could protect infants who have a genetic profile linked with asthma risk

Infants who have a genetic profile linked with asthma risk could be protected against respiratory symptoms if they are breastfeed, according to a new study. The study is presented today (4 September, 2016) at the European Respiratory Society's International Congress.

"Our study is the first to show that breastfeeding can modify the effect of asthma-related genetic profiles on respiratory symptoms in the first year of life", commented Dr Olga Gorlanova, from the University Children's Hospital Basel (UKBB), and the University of Basel, Basel, Switzerland.

Genes that are associated with asthma risk are located on chromosome 17 and called 17q21. A recent study reported that children who possessed genetic variants on chromosome 17q21 had an increased risk of developing wheeze, when combined with certain environmental exposures.

It is already known that environmental factors have a modifying effect on specific genetic risk, so the aim of this new study was to find out whether this could also be true for breastfeeding and this specific gene related to asthma with the respect to respiratory symptoms in early infancy.

368 infants from the Basel-Bern Infant Lung Development birth cohort in Switzerland were included in the study. Researchers collected data on occurrence and severity of respiratory symptoms, breastfeeding status and genotyping was performed.

Findings revealed that during the weeks that infants were breastfed, those carrying the asthma risk genotypes, had a 27% decreased relative risk of developing respiratory symptoms. When infants were not breastfed, those carriers exhibited a trend towards an increased risk of respiratory symptoms.

Dr Gorlanova said: "As research in this field progresses, we are understanding more and more about the gene-environment interaction for the development of asthma. Our study sheds light on how this interaction can be modified by breastfeeding. This is the first time that we were able to show the effect of the 17q21 variants on respiratory symptoms during the 1st year of life, depending on breastfeeding status. Our results must be replicated in another cohort."

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This Tiny Ankylosaur Ate... Fish?

Paleontologists claim to have found a little armored dinosaur that went carnivore

By Brian Switek

***Liaoningosaurus slurping fish.* Credit: Ji et al., 2016.**

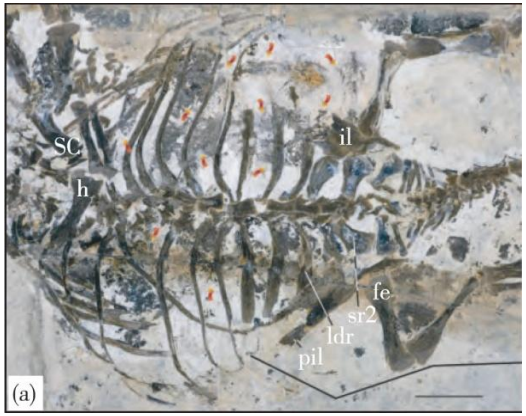
Ankylosaurs are weird. I'm not just talking about the lavish displays of osteoderms jutting every which way from their bodies. Look inside the skull of an ankylosaur, for example, and you may wonder how these armored dinosaurs fed themselves.

Their teeth were tiny and, while decent at cutting through plants, didn't allow them to chew. Now a new discovery has made one ankylosaur seem even stranger still. *Liaoningosaurus*, paleontologist Ji Qiang and colleagues write, may have fed on fish.

The dinosaur wasn't as imposing as the huge, club-tailed celebrities of its kind, like *Ankylosaurus* itself. *Liaoningosaurus paradoxus* lived much earlier, around 125-121 million years ago in what is now China, and the largest of their species were a little more than a foot long. Up until now, they were thought to be herbivores like the rest of their family. But now Ji and coauthors report that one *Liaoningosaurus* appears to have a belly full of fish.

The paleontologists consider three different ways this association might have come to be. Maybe the little dinosaur died and came to rest on top of a mass of fish that had already settled to the bottom. It could be coincidence. Then again, Ji and coauthors write, maybe the fish were sheltering inside the dead dinosaur when they, too, perished. But the favored interpretation in the new paper is that *Liaoningosaurus* was a dinosaur equivalent of a turtle, swimming around and snaffling up little fish.

A fish-eating ankylosaur isn't as outlandish as it might first sound. After all, as blog neighbor Darren Naish has often pointed out, there are herbivorous animals today that occasionally eat meat. Ji and colleagues are proposing something a little different for *Liaoningosaurus* - that the dinosaur was adapted to swimming around after piscine prey - but, even so, it's somewhat surprising that paleontologists haven't yet found evidence of a veggisaur having a cheat day.



***The little* Liaoningosaurus. *Red arrows mark fish remains.* Credit: Ji et al., 2016.**

But is this new *Liaoningosaurus* specimen the evidence of a plant-eating dinosaur gone carnivore we've been waiting for? Maybe not. Even though Ji and coauthors say that the coincidence of the dinosaur being buried with the fish is too "speculative", neither do they refute the possibility. The fish are in pieces, yes, but they're also scattered through the body cavity instead of being constrained to the gut. Additional evidence - such as acid etching on the fish and a CT scan showing the relationship of all the fossiliferous pieces - would be needed to confirm that *Liaoningosaurus* was doing something totally different from other ankylosaurs.

Paleontology is fueled by the yearning to envision prehistoric *life*. Yet the discipline is based on looking at scenes of death and burial. The place an organism is interred may not represent its natural habitat, and the way bodies are covered up may wash in other organisms that the species had no interaction with in life. To examine a saurian's life, we have to first recognize that we're starting with the story of its afterlife.

Ji, Q., Wu, X., Cheng, Y., Ten, F., Wang, X., Ji, Y. Fish hunting ankylosaurs (Dinosauria, Ornithischia) from the Cretaceous of China. Journal of Geology. *doi: 10.3969 /j.issn.1674 – 3636.2016.02.183*

http://bit.ly/2bNctkI

There is now a sixth taste – and it explains why we love carbs

Evidence that carbohydrate-rich foods may elicit a unique taste too

By Jessica Hamzelou

As any weight-watcher knows, carb cravings can be hard to resist. Now there's evidence that carbohydrate-rich foods may elicit a unique taste too, suggesting that "starchy" could be a flavour in its own right.

It has long been thought that our tongues register a small number of primary tastes: salty, sweet, sour and bitter. Umami – the savoury taste often associated with monosodium glutamate – was added to this list seven years ago, but there's been no change since then.

However, this list misses a major component of our diets, says Juyun Lim at Oregon State University in Corvallis. "Every culture has a major source of complex carbohydrate. The idea that we can't taste what we're eating doesn't make sense," she says.

Floury flavour

Complex carbohydrates such as starch are made of chains of sugar molecules and are an important source of energy in our diets. However, food scientists have tended to ignore the idea that we might be able to specifically taste them, says Lim. Because enzymes in our saliva break starch down into shorter chains and simple sugars, many have assumed we detect starch by tasting these sweet molecules.

Her team tested this by giving a range of different carbohydrate solutions to volunteers – who it turned out were able to detect a starch-like taste in solutions that contained long or shorter carbohydrate chains. "They called the taste 'starchy'," says Lim. "Asians would say it was rice-like, while Caucasians described it as bread-like or pasta-like. It's like eating flour."

The volunteers could still make out this floury flavour when they were given a compound that blocks the receptors on the tongue for detecting sweet tastes. This suggests we can sense carbohydrates before they have been completely broken down into sugar molecules.

When the volunteers were given a compound to block the salivary enzyme that breaks long chains of carbohydrate into shorter ones, they stopped sensing the taste of starch when given solutions containing only long-chain carbohydrates. This suggests that the floury flavour comes from the shorter chains.

This is the first evidence that we can taste starch as a flavour in its own right, says Lim.

Michael Tordoff at Monell Chemical Senses Center in Philadelphia is convinced by the evidence, and says it is impressive. "It will surprise a lot of people," he says.

Taste test

The finding adds to growing evidence that human taste is more complex than thought. "Many people think there are only five tastes, but a bunch of us think there might be others," says Tordoff, who is investigating whether we might be able to specifically taste calcium.

Other potential tastes being investigated are the flavour of carbonated drinks, the metallic taste you get from blood, and amino acids, the building blocks of proteins. Receptors have been found for kokumi, a full-bodied flavour that has been described as "hearty" and is thought to make foods feel richer and more satisfying, and there is some evidence that we can taste the fatty acids that make up fats. "We are moving away from the idea of five primary tastes," says Lim.

But before any new flavours can be enshrined as primary tastes, they must meet a strict list of criteria. Tastes need to be recognisable, have their own set of tongue receptors, and trigger some kind of useful physiological response.

Starch doesn't tick all of these boxes yet: Lim and her colleagues are yet to identify specific starch receptors on the tongue. Kokumi has not so far made the list because people who eat it can't put their finger on a specific taste.

One criterion is that a flavour must be useful to us. There's a strong case to be made for starch here, which is a valuable slow-release energy source that is worth detecting.

"I believe that's why people prefer complex carbs," says Lim. "Sugar tastes great in the short term, but if you're offered chocolate and bread, you might eat a small amount of the chocolate, but you'd choose the bread in larger amounts, or as a daily staple."

Journal reference: Chemical Senses, DOI: 10.1093/chemse/bjw088

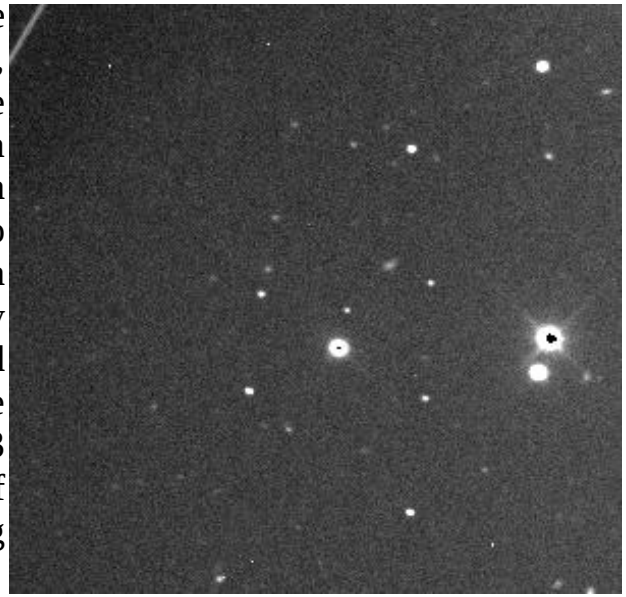
http://bit.ly/2cm52Ck

Freddie Mercury received an asteroid for his 70th birthday

'A shooting star leaping through the sky'

By Amar Toor @amartoo Sep 5, 2016, 5:27a

Freddie Mercury would have turned 70 years old today, and to mark the occasion, the late Queen frontman received his very own asteroid. In a YouTube video published on Sunday, Queen guitarist Brian May announced that Asteroid 17473 will from now on be known as Asteroid 17473 Freddiemercury, in honor of "Freddie's outstanding influence in the world."



Asteroid 17473 Freddiemercury

The announcement was formalized yesterday with a certificate of adoption from the International Astronomical Union and the Minor Planet Center. The asteroid, discovered in 1991, is located in the main Asteroid Belt between the orbits of Jupiter and Mars. Nearly two miles wide, the asteroid only reflects about 30 percent of the light that falls on it, and can only be seen with powerful telescopes; but it's an appropriate gift for the man who once sang of himself as "a shooting star leaping through the sky."

"It's just a dot of light," May said in the video, "but it's a very special dot of light."