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New antibiotic packs a punch against bacterial resistance *Advance could eliminate the threat of antibiotic-resistant infections for years to come*

LA JOLLA, CA - Scientists at The Scripps Research Institute (TSRI) have given new superpowers to a lifesaving antibiotic called vancomycin, an advance that could eliminate the threat of antibiotic-resistant infections for years to come. The researchers, led by Dale Boger, co-chair of TSRI's Department of Chemistry, discovered a way to structurally modify vancomycin to make an already-powerful version of the antibiotic even more potent.

"Doctors could use this modified form of vancomycin without fear of resistance emerging," said Boger, whose team announced the finding today in the journal Proceedings of the National Academy of Sciences. The original form of vancomycin is an ideal starting place for developing better antibiotics. The antibiotic has been prescribed by doctors for 60 years, and bacteria are only now developing resistance to it. This suggests bacteria already have a hard time overcoming vancomycin's original "mechanism of action," which works by disrupting how bacteria form cell walls.

Boger called vancomycin "magical" for its proven strength against infections, and previous studies by Boger and his colleagues at TSRI had shown that it is possible to add two modifications to vancomycin to make it even more potent. "With these modifications, you need less of the drug to have the same effect," Boger said.

The new study shows that scientists can make a third modification--which interferes with a bacterium's cell wall in a new way--with promising results. Combined with the previous modifications, this alteration gives vancomycin a 1,000-fold increase in activity, meaning doctors would need to use less of the antibiotic to fight infection.

The discovery makes this version of vancomycin the first antibiotic to have three independent mechanisms of action. "This increases the durability of this antibiotic," said Boger. "Organisms just can't

simultaneously work to find a way around three independent mechanisms of action. Even if they found a solution to one of those, the organisms would still be killed by the other two."

Tested against Enterococci bacteria, the new version of vancomycin killed both vancomycin-resistant Enterococci and the original forms of Enterococci.

The next step in this research is to design a way to synthesize the modified vancomycin using fewer steps in the lab, as the current method takes 30 steps. But Boger calls this the "easy part" of the project, compared with the challenge of designing the molecule in the first place.

Even if the process isn't streamlined, Boger believes the new vancomycin's lifesaving powers make its production valuable. "Antibiotics are total cures for bacterial infections," said Boger. "Making this molecule is important, even by the current approach, if the failure of antibiotics continues."

In addition to Boger, authors of the study, "Peripheral modifications of [Ψ [CH₂NH]Tpg₄]vancomycin with added synergistic mechanisms of action provide durable and potent antibiotics," included first author Akinori Okano and Nicholas A. Isley, both of TSRI.

The study was supported by the National Institutes of Health (grants F32 GM114948 and CA041101).

<http://bit.ly/2rEuyxA>

How the visual cortex changes from birth to old age *A study of post-mortem brain tissue reveals the human primary visual cortex (V1) develops gradually throughout life.*

The research, published in The Journal of Neuroscience, may help to explain why the structure of this part of the brain matures in the first years after birth while vision continues to change throughout the lifespan.

Kathryn Murphy and colleagues obtained brain samples from 30 deceased individuals ranging from birth to 80 years of age to study how expression of a set of glutamatergic proteins (which regulate neurotransmission at a majority of synapses in human V1) changes in

this region over time. The results show development of human V1 occurs in five different stages that mirror changes in vision. For example, the expression of three of these proteins peaks between 5 and 11 years of age, which coincides with the end of the period when children are susceptible to developing amblyopia, or lazy eye. Another protein did not peak until about 40 years of age and then dropped dramatically, by about 75 percent, in adults over 55 years of age, perhaps signaling degeneration in human V1.

The authors conclude that their work may inform the development of vision therapies for individuals of different ages.

<http://bit.ly/2qBqS26>

CRISPR gene editing can cause hundreds of unintended mutations

Gene-editing technology can introduce hundreds of unintended mutations into the genome

New York, NY - As CRISPR-Cas9 starts to move into clinical trials, a new study published in Nature Methods has found that the gene-editing technology can introduce hundreds of unintended mutations into the genome.

"We feel it's critical that the scientific community consider the potential hazards of all off-target mutations caused by CRISPR, including single nucleotide mutations and mutations in non-coding regions of the genome," says co-author Stephen Tsang, MD, PhD, the Laszlo T. Bito Associate Professor of Ophthalmology and associate professor of pathology and cell biology at Columbia University Medical Center and in Columbia's Institute of Genomic Medicine and the Institute of Human Nutrition.

CRISPR-Cas9 editing technology -- by virtue of its speed and unprecedented precision -- has been a boon for scientists trying to understand the role of genes in disease. The technique has also raised hope for more powerful gene therapies that can delete or repair flawed genes, not just add new genes.

The first clinical trial to deploy CRISPR is now underway in China, and a U.S. trial is slated to start next year. But even though CRISPR can precisely target specific stretches of DNA, it sometimes hits other parts of the genome. Most studies that search for these off-target mutations use computer algorithms to identify areas most likely to be affected and then examine those areas for deletions and insertions.

"These predictive algorithms seem to do a good job when CRISPR is performed in cells or tissues in a dish, but whole genome sequencing has not been employed to look for all off-target effects in living animals," says co-author Alexander Bassuk, MD, PhD, professor of pediatrics at the University of Iowa.

In the new study, the researchers sequenced the entire genome of mice that had undergone CRISPR gene editing in the team's previous study and looked for all mutations, including those that only altered a single nucleotide.

The researchers determined that CRISPR had successfully corrected a gene that causes blindness, but Kellie Schaefer, a PhD student in the lab of Vinit Mahajan, MD, PhD, associate professor of ophthalmology at Stanford University, and co-author of the study, found that the genomes of two independent gene therapy recipients had sustained more than 1,500 single-nucleotide mutations and more than 100 larger deletions and insertions. None of these DNA mutations were predicted by computer algorithms that are widely used by researchers to look for off-target effects.

"Researchers who aren't using whole genome sequencing to find off-target effects may be missing potentially important mutations," Dr. Tsang says. "Even a single nucleotide change can have a huge impact."

Dr. Bassuk says the researchers didn't notice anything obviously wrong with their animals. "We're still upbeat about CRISPR," says Dr. Mahajan. "We're physicians, and we know that every new therapy has some potential side effects--but we need to be aware of what they are."

Researchers are currently working to improve the components of the CRISPR system--its gene-cutting enzyme and the RNA that guides the enzyme to the right gene--to increase the efficiency of editing.

"We hope our findings will encourage others to use whole-genome sequencing as a method to determine all the off-target effects of their CRISPR techniques and study different versions for the safest, most accurate editing," Dr. Tsang says.

The paper is titled, "Unexpected mutations after CRISPR-Cas9 editing in vivo." Additional authors are Kellie A. Schafer (Stanford University), Wen-Hsuan Wu (Columbia University Medical Center), and Diana G. Colgan (Iowa).

The research was supported by the National Institutes of Health (grants R01NS098590, R01EY026682, R01EY024665, R01EY025225, R01EY024698, R21AG050437, 5P30EY019007, R01EY018213, 5P30CA013696, and F31EY026789), Jonas Children's Vision Care, Research to Prevent Blindness, the Tistou and Charlotte Kerstan Foundation, the Schneeweiss Stem Cell Fund, New York State (grant CO29572), the Crowley Family Fund, and the Gebroe Family Foundation.

The authors declare no financial or other conflicts of interest.

<http://bit.ly/2seeaRv>

Rice was domesticated in China almost 10,000 years ago
Carbon dating of rice fossils from Shangshan in the Lower Yangtze region shows domestication occurred there at least 9,400 years ago,
writes Amy Middleton.

The earliest known domestic rice was cultivated in China as early as 9,400 years ago, at the beginning of the Holocene epoch, according to a new study.

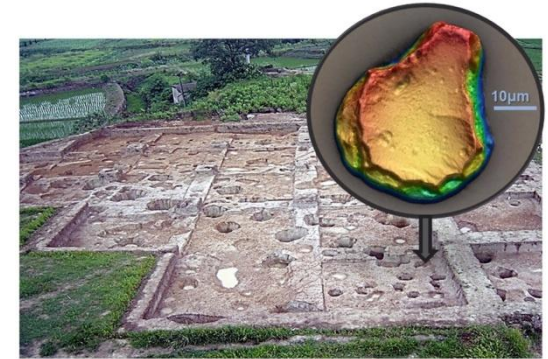
The site of the first domestication of rice is a [hotly contested issue](#), with several countries eager to lay claim. Several locations in China have been put forward, as have the Ganges valley in India, the southern slopes of the Himalayas, and various places in southeast Asia. There is even evidence to suggest that [wild rice may have originated in Australia](#).

Shangshan, in China's Lower Yangtze region, has long been one of the strongest contenders: fossils uncovered there are some of the

earliest evidence of rice grown by humans. Remnants of rice are identified through microscopic bodies of silica called phytoliths. However, the species of rice – whether wild or domesticated – isn't always clear from these remains.

Furthermore, although researchers have used radiocarbon dating to place these phytoliths on a historic timeline, their estimates have been controversial – where other materials are also present, as is the case on pottery and tools, fossils can be contaminated, interrupting the dating process.

The Shangshan site and a rice bulliform phytolith used for dating and identification (inset). Houyuan Lu



In this study, a research team led by Xinxin Zuo, a geophysicist at the Chinese Academy of Sciences in Beijing, verified the age of the phytoliths at Shangshan by comparing them with carbon dating on other materials from the same environment.

The team then examined the morphological characteristics of the rice remnants, and confirmed that the species is closer to modern-day rice than wild rice.

Their results, [published in the Proceedings of the National Academy of Science](#), suggest the Shangshan fossils are indeed evidence of the earliest domesticated rice, dating back to 9,400 years ago.

"When the domestication of rice began in its homeland, China, is an enduring and important issue of debate for researchers from many different disciplines," the researchers write.

"Our results indicate that rice domestication may have begun at Shangshan in the Lower Yangtze during the beginning of the Holocene."

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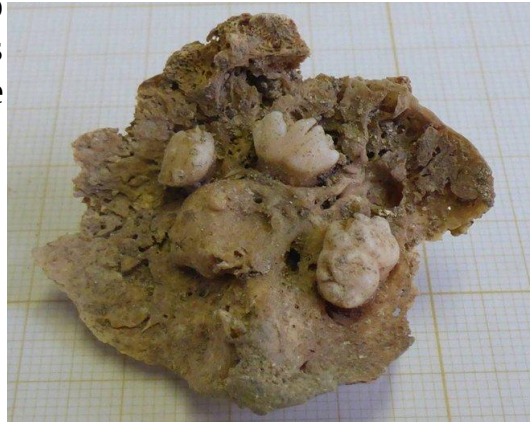
A Tumor with Teeth Discovered in Gothic Graveyard *Archaeologists excavating a gothic church graveyard in Lisbon, Portugal, made a discovery for the annals of medical history: an ovarian tumor that had started forming teeth.*

By Megan Gannon, Live Science Contributor | May 30, 2017 06:58am

Today, doctors know that this type of cyst, called a teratoma, is the most common tumor that occurs in the ovaries.

But scientists are just starting to learn about past teratoma cases thanks to new evidence from the archaeological record.

A [teratoma](#), which essentially translates as "monstrous swelling" from Greek, can occur when cells that should become eggs start multiplying abnormally and form mature tissues like hair, teeth and bones.



Archaeologists discovered this ovarian teratoma, or a tumor that had started sprouting teeth, in a burial outside the Church and Convent of Carmo in Lisbon.

Sofia N. Wasterlain et al./[International Journal of Paleopathology](#)

These cysts account for up to 20 percent of all ovarian tumors, and most develop in women of reproductive age, according to past [studies](#). These masses are usually benign and go unnoticed, without causing any symptoms. But some can be [cancerous](#), and some can grow so large that they cause severe pain, or twisting in the ovaries. The largest reported teratoma was 18 inches by 10 inches (45 by 25 centimeters), removed from a 74-year-old woman, according to [one review](#).

While many teratomas look like balls of tissue, some can develop so much that they take the shape of a fetus. In 2004, doctors in Japan reported the [discovery of a "doll-like" teratoma](#) with a head and limbs in the ovary of a 25-year-old virginal woman.

The tumor newly unearthed in Portugal measures 1.7 inches (4.3 cm) at its widest point, according to a study published May 12 in the [International Journal of Paleopathology](#). The mass is embedded with at least five malformed teeth, and it shows signs of some disorganized bone formation.

Researchers discovered the tumor during the excavation of 42 burials outside the Church and Convent of Carmo in Lisbon in 2010 and 2011. The calcified mass was resting near the pelvic area of a woman who was over 45 years old at the time of her death, the study said. This cemetery was used from the early 15th century until the devastating 1755 earthquake that wrecked the church and many other buildings in Lisbon, so the researchers assume the woman lived sometime during that era, the study said.

"When the archaeologists found this ovarian mass, obviously they immediately noticed they were in the presence of a very unusual thing that should be carefully recovered and transported for further analysis in the laboratory," study leader Sofia Wasterlain of Portugal's University of Coimbra told Live Science. "However, at that time they didn't know what it was exactly."

Wasterlain and her colleagues considered other explanations for this little bony ball, such as a dead fetus or an ectopic pregnancy (where the embryo attaches outside the uterus) that calcified within the woman's body. But they concluded that this case looks most like [a teratoma](#). It's not possible to tell if the tumor had any effect on the life or death of the woman, but her skeleton didn't seem to have any changes related to the tumor, the report said.

"Some types of tumors that are thought to be characteristic of modern societies and commonly attributed to Western civilization are also found in past populations," the researchers wrote in the study. "This case also draws attention to the importance of conducting meticulous archaeological excavation in order to preserve rare, but significant findings. During excavation of human remains, materials from body

cavities, which may provide clues not directly accessible from the skeleton, should always be sought and recovered with care."

This case in Portugal is not the first time a teratoma like this has been unearthed in a graveyard. In 2013, archaeologists digging at a Roman necropolis in Spain reported that they found the 1,600-year-old remains of a woman who had a [calcified tumor in her pelvis](#).

<http://bit.ly/2qIk1Nz>

Volcanic 'geoengineering' may have caused a climate catastrophe that killed most animal species

Death by volcano?

Anyone concerned by the idea that people might try to combat global warming by injecting tons of sulfate aerosols into Earth's atmosphere may want to read an article in the May 1, 2017 issue of the journal *Geology*.

In it, a Washington University in St. Louis scientist and his colleagues describe what happened when pulses of atmospheric carbon dioxide and sulfate aerosols were intermixed at the end of the Ordovician geological period more than 440 millions years ago.

The counterpart of the tumult in the skies was death in the seas. At a time when most of the planet north of the tropics was covered by an ocean and most complex multicellular organisms lived in the sea, 85 percent of marine animal species disappeared forever. The end Ordovician extinction, as this event was called, was one of the five largest mass extinctions in Earth's history.

Although the gases were injected into the atmosphere by massive volcanism rather than prodigious burning of fossil fuels and under circumstances that will never be exactly repeated, they provide a case history that reveals the potential instability of planetary-scale climate dynamics.

Figuring out what caused the end Ordovician extinction or any of the other mass extinctions in Earth's history is notoriously difficult, said David Fike, associate professor of earth and planetary sciences in Arts & Sciences and a co-author on the paper.

Because the ancient atmospheres and oceans have long since been altered beyond recognition, scientists have to work from proxies, such as variations in oxygen isotopes in ancient rock, to learn about climates long past.

The trouble with most proxies, said Fike, who specializes in interpreting the chemical signatures of biological and geological activity in the rock record, is that most elements in rock participate in so many chemical reactions that a signal can often be interpreted in more than one way.

But a team led by David Jones, an earth scientist at Amherst College, was able to bypass this problem by measuring the abundance of mercury. Today, the primary sources of mercury are coal-burning power plants and other anthropogenic activities; during the Ordovician, however, the main source was volcanism.

Volcanism coincides with mass extinctions with suspicious frequency, Fike said. He is speaking not about an isolated volcano but rather about massive eruptions that covered thousands of square kilometers with thick lava flows, creating large igneous provinces (LIPs). The most famous U.S. example of a LIP is the Columbia River Basalt province, which covers most of the southeastern part of the state of Washington and extends to the Pacific and into Oregon.

Volcanoes are plausible climate forcers, or change agents, because they release both carbon dioxide that can produce long-term greenhouse warming and sulfur dioxide that can cause short-term reflective cooling.

In addition, the weathering of vast plains of newly exposed rock can draw down atmospheric carbon dioxide and bury it as limestone minerals in the oceans, also causing cooling.

When Jones analyzed samples of rock of Ordovician age from south China and the Monitor Range in Nevada, he found anomalously high mercury concentrations. Some samples held 500 times more mercury than the background concentration. The mercury arrived in three pulses, before and during the mass extinction.

But what happened? It had to have been an unusual sequence of events because the extinction (atypically) coincided with glaciation and also happened in two pulses.

As the scientists began to piece together the story, they began to wonder if the first wave of eruptions didn't push Earth's climate into a particularly vulnerable state, setting it up for a climate catastrophe triggered by later eruptions.

The first wave of eruptions laid down a LIP whose weathering then drew down atmospheric carbon dioxide. The climate cooled and glaciers formed on the supercontinent of Gondwana, which was then located in the southern hemisphere.

The cooling might have lowered the tropopause, the boundary between two layers of the atmosphere with different temperature gradients. The second wave of volcanic eruptions then injected prodigious amounts of sulfur dioxide above the tropopause, abruptly increasing the Earth's albedo, or the amount of sunlight it reflected.

This led to the first and largest pulse of extinctions. As ice sheets grew, sea level dropped and the seas became colder, causing many species to perish.

During the second wave of volcanism, the greenhouse warming from carbon dioxide overtook the cooling caused by sulfur dioxide and the climate warmed, the ice melted and sea levels rose. Many of the survivors of the first pulse of extinctions died in the ensuing flooding of habitat with warmer, oxygen-poor waters.

The take-home, said Fike, is that the different factors that affect Earth's climate can interact in unanticipated ways and it is possible that events that might not seem extreme in themselves can put the climate system into a precarious state where additional perturbations have catastrophic consequences.

"It's something to keep in mind when we contemplate geoengineering schemes to mitigate global warming," said Fike, who teaches a course where students examine such schemes and then evaluate their willingness to deploy them.

<http://bit.ly/2qO6WSq>

Emergency room patients routinely overcharged, study finds

'Price gouging' is worst for minorities and uninsured

An analysis of billing records for more than 12,000 emergency medicine doctors across the United States shows that charges varied widely, but that on average, adult patients are charged 340 percent more than what Medicare pays for services ranging from suturing a wound to interpreting a head CT scan.

A report of the study's findings, published in *JAMA Internal Medicine* on May 30, also notes that the largest hospitals markups are more likely made to minorities and uninsured patients.

"There are massive disparities in service costs across emergency rooms and that price gouging is the worst for the most vulnerable populations," says [Martin Makary, M.D., M.P.H.](#), professor of surgery at the Johns Hopkins University School of Medicine and the study's senior investigator. "This study adds to the growing pile of evidence that to address the huge disparities in health care, health care pricing needs to be fairer and more transparent," adds Makary, whose widely published research focuses on health care costs and disparities.

For the study, Makary and his team obtained Medicare billing records for 12,337 emergency medicine physicians practicing in nearly 300 hospitals all 50 states in 2013 to determine how much emergency departments billed for services compared to the Medicare allowable amount.

The Medicare allowable amount is the sum of what Medicare pays, the deductible and coinsurance that patients pay, and the amount any third party such as the patient pays.

In addition, using the 2013 American Hospital Association database, the research team identified size, urban/rural status, teaching status, for-profit status, regional location and safety-net hospital status for each emergency medicine department whose billing data were made part of the analysis. Using the zip code for each emergency

department, the researchers also estimated poverty rates, uninsured status and minority populations for those using each emergency room, based on data from the 2013 U.S. Census Bureau.

The researchers then calculated each service bill's markup ratio, defined as the relationship between the billed charges and the Medicare allowable amount. For example, a markup ratio of 4.0 means that for a service with a Medicare allowable amount of \$100, the hospital charged \$400, or 300% over the Medicare allowable amount.

Makary and his team found that emergency departments charged anywhere from 1.0-12.6 times (\$100-\$12,600) more than what Medicare paid for services. On average, emergency medicine doctors had a markup ratio of 4.4 (340 percent in excess charges), or emergency medicine physician charges of \$4 billion versus \$898 million in Medicare allowable amounts.

The researchers also analyzed billing information for 57,607 general internal medicine physicians 3,669 hospitals in all 50 states to determine whether any markup differences, and how much, existed between emergency medicine physicians practicing in a hospital's ER, and general internal medicine physicians who see patients at hospitals. On average, charges were greater when a service was performed by an emergency medicine physician rather than a general internal medicine physician. Overall, general internal medicine physicians had an average markup ratio of 2.1 compared to the Medicare allowable amount.

Makary found that wound closure had the highest median markup ratio at 7.0, and interpreting head CT scans had the greatest within-hospital variation, with markup ratios ranging between 1.6 and 27.

For a physician interpretation of an electrocardiogram, the median Medicare allowable rate is \$16, but different emergency departments charged anywhere from \$18 to \$317, with a median charge of \$95 (or a markup ratio of 6.0). General internal medicine doctors in hospitals charged an average of \$62 for the same service.

Overall, emergency departments that charged patients the most were more likely to be located in for-profit hospitals in the southeastern and Midwestern U.S., and served higher populations of uninsured, African-American and Hispanic patients.

Our study found that inequality is then further compounded on poor, minority groups, who are more likely to receive services from hospitals that charge the most," says Makary.

While the study was limited by lack of data on facility and technical fees also charged by the hospital, as well as lack of patients' insurance type and the actual amount patients ultimately paid, Makary says the study highlights the urgent need for legislation that will protect uninsured patients.

"This is a health care systems problem that requires state and federal legislation to protect patients. New York has passed a law that requires hospital and insurance companies to agree on a cost for the care so patients are not billed egregious amounts. Patients really have no way of protecting themselves from these pricing practices," says Tim Xu, a fourth year medical student at the Johns Hopkins University School of Medicine and the paper's first author.

Models such as the Maryland Waiver, Makary adds, where prices are set at the same rate no matter what hospital a patient goes to, can increase price transparency and protect patients. Currently, at least 7 states have passed some legislation to protect uninsured patients from paying so called charge master prices, a list of billable services developed and closely guarded by each hospital, noting prices that are usually highly inflated and charged mainly to uninsured and other "self pay" patients. However, Makary says a national model is necessary to unveil what is currently an inexplicably chaotic and opaque pricing system.

Other authors on this paper include Angela Park, Sarah Joo, Susan Hutflless and Ambar Mehta of The Johns Hopkins University.

<http://bit.ly/2qKLUJ4>

Handwashing: Cool water as effective as hot for removing germs

Rutgers study indicates that washing for 10 seconds eliminates harmful bacteria

We all know that washing our hands can keep us from spreading germs and getting sick. But a new Rutgers-New Brunswick study found that cool water removes the same amount of harmful bacteria as hot.

"People need to feel comfortable when they are washing their hands but as far as effectiveness, this study shows us that the temperature of the water used didn't matter," said Donald Schaffner, distinguished professor and extension specialist in food science.

In the Rutgers study, published in the June issue of the Journal of Food Protection, high levels of a harmless bacteria were put on the hands of 21 participants multiple times over a six-month period before they were asked to wash their hands in 60-degree, 79-degree or 100-degree water temperatures using 0.5 ml, 1 ml or 2 ml volumes of soap.

"This study may have significant implications towards water energy, since using cold water saves more energy than warm or hot water," said Schaffner. "Also we learned even washing for 10 seconds significantly removed bacteria from the hands."

While the study indicates that there is no difference between the amount of soap used, more work needs to be done to understand exactly how much and what type of soap is needed to remove harmful microbes from hands, said co-author Jim Arbogast, vice president of Hygiene Sciences and Public Health Advancements for GOJO. "This is important because the biggest public health need is to increase handwashing or hand sanitizing by foodservice workers and the public before eating, preparing food and after using the restroom," Arbogast said.

These findings are significant, particularly to the restaurant and food industry, because the U.S. Food and Drug Administration issues

guidelines, every four years, to states. Those guidelines currently recommend that plumbing systems at food establishments and restaurants deliver water at 100 degrees Fahrenheit for handwashing. Schaffner said the issue of water temperature has been debated for a number of years without enough science to back-up any recommendation to change the policy guidelines or provide proof that water temperature makes a difference in hand hygiene. Many states, in fact, interpret the FDA guidelines as a requirement that water temperature for handwashing must be 100 degrees, he said.

The FDA is scheduled to hold a conference in 2018 to discuss the existing code and any modifications that should be made and Schaffner would like to see the water temperature policy revised at that time.

"I think this study indicates that there should be a policy change," said Schaffner. "Instead of having a temperature requirement, the policy should only say that comfortable or warm water needs to be delivered. We are wasting energy to heat water to a level that is not necessary."

<http://bit.ly/2qPvaeV>

Diabetes linked to bacteria invading the colon, study finds

Metabolic disease is correlated with having bacteria that penetrate the mucus lining of the colon

ATLANTA--In humans, developing metabolic disease, particularly type 2 diabetes, is correlated with having bacteria that penetrate the mucus lining of the colon, according to a study led by Drs. Benoit Chassaing and Andrew Gewirtz at Georgia State University.

The findings, which provide insight on how people develop insulin resistance-associated dysglycemia (abnormal blood glucose levels), are published in the journal Cellular and Molecular Gastroenterology and Hepatology.

Metabolic syndrome is the term for a group of factors that raise a person's risk for heart disease and other health problems, such as diabetes and stroke. Such risk factors include a large waistline, a high triglyceride level (type of fat found in the blood), low HDL

cholesterol level, high blood pressure and high fasting blood sugar levels. Metabolic syndrome, which has become far more common due to a rise in obesity rates among adults, is a leading risk factor for many serious, life-threatening diseases, including type 2 diabetes and heart disease, according to the National Institutes of Health.

"Alterations in bacteria have been associated with metabolic diseases, including obesity and type 2 diabetes, but mechanisms remain elusive," said Gewirtz, professor in the Institute for Biomedical Sciences at Georgia State.

"Previous studies in mice have indicated that bacteria that are able to encroach upon the epithelium might be able to promote inflammation that drives metabolic diseases, and now we've shown that this is also a feature of metabolic disease in humans, specifically type 2 diabetics who are exhibiting microbiota encroachment."

The epithelium is the mucus-lined cellular covering of internal and external surfaces of the body, including the intestinal tract. Gut microbiota is the collective term for the communities of microscopic living organisms that inhabit this environment. Gut microbiota that live in the outer regions of the mucus and remain a safe distance from epithelial cells provide a benefit to the host, but Chassaing and Gewirtz hypothesize that microbiota that encroach upon host cells drive chronic inflammation that interferes with the normal action of insulin, promoting type 2 diabetes.

In this study, the researchers used samples from human subjects enrolled at the Veteran's Administration Hospital in Atlanta. The subjects, at least 21 years old with no major health problems besides diabetes, were undergoing colonoscopy for colon cancer screening. The researchers obtained each subject's history of diabetes and gastrointestinal complaints through interviews and reviewing medical records. During the colonoscopy procedure, two mucosal biopsies were taken from the left colon and analyzed.

The study's results have impressed leading experts in diabetes research.

"The data are impressive and may have opened a new field of investigation in metabolic function and type 2 diabetes," said Dr. Samuel Klein, chief of the Division of Geriatrics and Nutritional Science at the Washington University School of Medicine Diabetes Research Center.

The researchers are conducting follow-up studies to determine the identity of the bacteria that are invading the colon lining and are exploring remedies to prevent such bacteria encroachment.

Chassaing, assistant professor in the Institute for Biomedical Sciences and Center for Inflammation, Immunity and Infection, is lead author of the study.

Co-authors of the paper include Dr. Shreya M. Raja and Dr. Shanthi Srinivasan of Emory University School of Medicine and Dr. James D. Lewis of Perelman School of Medicine at University of Pennsylvania.

The study is funded by the National Institutes of Health and the Crohn's and Colitis Foundation.

To read the paper, visit [http://www.cmghjournal.org/article/S2352-345X\(17\)30075-9/fulltext](http://www.cmghjournal.org/article/S2352-345X(17)30075-9/fulltext).

<http://bit.ly/2syMaaq>

Why You Should Chew Gum Until You Fart After a C-Section

Among the difficult things a woman has to do after having a C-section — from tending to her stitches to learning to breastfeed — now there's a recommendation that's a little easier: chew gum.

By Sara G. Miller, Staff Writer

A new meta-analysis suggests that chewing gum three times a day for 30 minutes each time can help bring back women's normal gut function after a [C-section delivery](#).

Up to one in five women develop a condition called "postoperative ileus" after a C-section, according to the meta-analysis, published online May 14 in [The Journal of Maternal-Fetal & Neonatal Medicine](#). "Postoperative ileus" means that the normal movements of a person's bowels — which squeeze and relax to move food along — slow down or sometimes even stop entirely, said senior study author Dr. Vincenzo Berghella, an OB-GYN at Thomas Jefferson University Hospital in Philadelphia.

This slowing down of gut movements can lead to symptoms including nausea, abdominal pain and bloating, Berghella told Live Science. Any type of abdominal surgery, not just a [C-section](#), can cause the condition, he added. It's thought to be caused by the inflammation that results from cutting open the abdomen.

Normally, postoperative ileus goes away on its own in about three or four days after an operation, but people can be uncomfortable while the condition lasts, the meta-analysis said.

Doctors can take several different approaches to help a woman get her gut moving again, including suggesting that the woman get up and walk around, or eat soon after the operation, Berghella said. But because women with postoperative ileus can feel nauseous, they may not want to eat, he noted.

Enter chewing gum.

[Chewing gum](#) can trick the body into thinking that the person is eating, Berghella said. It gets saliva flowing in the mouth and can help send signals to the gut to start moving again, he said.

In the meta-analysis, the researchers looked at 17 studies that included a total of more than 3,000 women. All of the studies focused on one simple question: How long did it take after the C-section for the woman to fart?

Passing gas is one of the earliest signs that a person's bowels are back to [functioning normally](#), Berghella said. It's a signal that there are no blockages in the gut and things are moving along, he added.

In most of the studies, the women were given gum within 2 hours of their delivery, and asked to chew it three times a day for 15 to 30 minutes at a time. The researchers found that, on average, women who chewed gum farted about 6.5 hours sooner than those who were not given gum: [Gum chewers](#) experienced their first fart about 23 hours after the operation, compared with non-chewers, who didn't fart until about 29.5 hours after the operation. There were no side effects from chewing gum, Berghella said.

Dr. Gabriele Saccone, an OB-GYN at the University of Naples Federico II in Italy and an author of the study, added that chewing gum after a C-section is a simple and inexpensive way for women to help get the gut moving again.

The new meta-analysis adds to a growing body of evidence suggesting that giving women gum after a C-section is a safe and effective way to help bring back gut function after the operation. A [2016 Cochrane review](#), for example, concluded that "gum chewing in the first 24 hours after a [C-section] is a well-tolerated, simple, low-cost, safe and easy intervention that enhances early recovery of bowel function, improves maternal comfort and potentially reduces hospital costs."

The researchers noted that more high-quality studies are needed to increase the evidence that supports giving women gum after a C-section.

But Berghella said he hopes that one day he can add "chew gum until you pass your first gas" to his postoperative orders to patients.

<http://bit.ly/2s6hiBC>

Living fossil challenges thinking on brain evolution
An ancient sea creature, discovered off the coast of Scotland in 2011, has shed new light on how evolution formed the modern brain.

An international team involving researchers at the University of St Andrews have examined the amphioxus, also known as the Lancelet, which was thought to be a brainless, faceless fish. Instead, they found it has a very complex brain which confounds previous understanding of how vertebrate brains evolved.



Credit: University of St Andrews

The latest research, carried out by the Universities of St Andrews, Murcia and Barcelona and the Centre for Genomic Regulation, published in *PLoS Biology*, compared the amphioxus brain with

current models of brain development in vertebrates, such as chicks and fish.

The new research casts doubt on the current textbook idea that the complex vertebrate brain evolved from a simple three-part brain composed of forebrain, midbrain and hindbrain. Rather, the new research suggests that the vertebrate brain must originally have formed from two parts.

Dr Ildiko Somorjai of the School of Biology at the University of St Andrews, co-author of the study, said: "Amphioxus is an amazing creature that can tell us a lot about how we have evolved. Humans have enormous brains with a large number of anatomical subdivisions to allow processing of complex information from the environment, as well as behavioural and motor control and language.

"Research in amphioxus tells us that even an outwardly simple brain may have complex regionalisation. It also strengthens the position of amphioxus as an important non-vertebrate model for understanding vertebrate evolution and development, with clear implications for biomedical research."

Described as a "brainless, faceless fish", amphioxus was found off the coast of Orkney during a marine survey in 2011, and is thought to be among the first animals to have evolved a structure similar to a backbone, the notochord.

Despite its appearance, amphioxus is not a [fish](#). It has a primitive spinal cord which runs down its back, but no clearly defined face, no bones or jaws and a small [brain](#) with a single light-sensing "frontal eye". It has changed so little for hundreds of millions of years that it has been described as a "living fossil".

As the best living proxy for the vertebrate ancestor, amphioxus gives important insight into what humanity's distant ancestor looked like, and how it might have behaved.

[New study revises the development and evolutionary origin of the vertebrate brain](#)

More information: Beatriz Albuixech-Crespo et al. Molecular regionalization of the developing amphioxus neural tube challenges major partitions of the vertebrate brain, *PLOS Biology* (2017). [DOI: 10.1371/journal.pbio.2001573](https://doi.org/10.1371/journal.pbio.2001573)

<http://bit.ly/2qOX9QQ>

Possible correlation shown between TMI nuclear accident and thyroid cancers

Penn State College of Medicine researchers have shown, for the first time, a possible correlation between the partial meltdown of the Three Mile Island Nuclear Generating Station and thyroid cancers in the counties surrounding the plant.

Three Mile Island (TMI), located near Harrisburg, Pennsylvania, had a partial meltdown accident on March 28, 1979. During the accident, radiation was released into the environment, which the United States Nuclear Regulatory Commission said was in small amounts with no detectable health effects.

Looking at tumor samples from people verified to have lived in the areas around TMI at the time of the accident, remained in the area and subsequently developed thyroid cancer, researchers observed a shift in cases to cancer mutations consistent with radiation exposure, from those consistent with random causes.

In this retrospective cohort study -- meaning the patients in the study already had thyroid cancer and were known to have been exposed to the TMI accident -- lead researcher David Goldenberg, professor of surgery, and colleagues identified 44 patients who were treated at the Penn State Milton S. Hershey Medical Center for the most common type of thyroid cancer -- papillary thyroid cancer -- between 1974 and 2014. The patients were then divided into two groups: at-risk and control groups.

Patients in the at-risk group were those who developed cancer between 1984 and 1996, consistent with known latency periods of radiation-induced thyroid cancer, and who lived in at-risk geographical areas -- based on reported weather patterns -- at the time of the accident.

"This definition was designed to allow us to identify relatively acute effects of radiation exposure from the accident," said Goldenberg.

Patients who developed cancer outside of the expected latency period were placed in the control group.

Researchers searched through all thyroid cancer tumor samples in the hospital's possession from the study period for patients who lived in at-risk regions Dauphin, York, eastern Cumberland, Lancaster and western Lebanon counties. They used genealogical software to verify that the patient was in an at-risk area during the accident, remained until cancer developed and was treated at the Medical Center. The tumor samples of those patients who were positively linked to the TMI accident area were then processed through the Penn State Institute for Personalized Medicine to determine genetic makeup of the cancer.

While most thyroid cancers are sporadic, meaning they happen without clear reasons, exposure to radiation has been shown to change the molecular makeup of the cancer, according to the researchers.

The researchers observed an increase in the genetic mutation caused by exposure to low-dose radiation in the at-risk group and a decrease in the incidence of sporadic thyroid cancer, identified by a specific genetic mutation known as BRAF. The BRAF mutation is typically not present in the radiation-induced types of thyroid cancer.

The study, which appeared in the May 29 supplement to the journal *Laryngoscope*, indicates that these observations are consistent with other radiation-exposed populations.

In the control group, 83 percent of patients had the BRAF mutation. The BRAF mutation was found in only 53 percent of patients in the at-risk group. In the at-risk group, there was also a rise in other molecular markers seen in radiation-induced thyroid cancer, the researchers added.

"While no single marker can determine whether an individual tumor is radiation-induced, these data support the possibility that radiation released from TMI altered the molecular profile of thyroid cancers in the population surrounding the plant," Goldenberg said.

A limitation of this study is the small sample size, limited to tumor samples from patients treated for thyroid cancer at Penn State Health Milton S. Hershey Medical Center. The next step in the research is a study with a larger number of patients from other regional hospitals to determine if the correlation continues in a larger sample.

"All patients were screened extensively to ensure that they lived in the vicinity of TMI from the date of the accident until they developed thyroid cancer," Goldenberg said. "We used an extensive vetting process to ensure that patients included in the study were present in at-risk counties at the time of the accident and to confirm, to the greatest extent possible, that patients resided in affected areas for their entire lives. Our study represents a static population, which increased our ability to detect radiation-induced cancers."

Past studies about thyroid cancer and TMI have showed variable results, mainly because they were demographic studies that looked at the entire population and not just those who met the criteria of the current study.

"Much of the variability associated with these studies is likely due to the relatively small size of the population surrounding the TMI plant relative to the large population required to detect statistically significant increases in cancer incidence following low-level radiation, combined with a high degree of mobility in the local population," Goldenberg said.

Other researchers who participated in this study include: Mariano Russo, biomedical sciences graduate program; Kenneth Houser, research technologist; Henry Crist, M.D., associate professor of pathology; Jonathan B. Derr, research technologist ; Vonn Walters, Ph.D., assistant professor of biochemistry and molecular biology; Joshua Warrick, M.D., assistant professor of pathology; Kathryn E. Sheldon, Ph.D., research project manager; James Broach, Ph.D., chair, biochemistry and molecular biology; Darrin V. Bann, M.D., Ph.D., surgery resident.

The George Laverty Foundation funded this research.

<http://bit.ly/2qUefrt>

Brain's immune cells linked to Alzheimer's, Parkinson's, schizophrenia

Scientists have, for the first time, characterized the molecular markers that make the brain's front lines of immune defense--cells called microglia--unique.

LA JOLLA - In the process, they discovered further evidence that microglia may play roles in a variety of neurodegenerative and psychiatric illnesses, including Alzheimer's, Parkinson's and Huntington's diseases as well as schizophrenia, autism and depression. "Microglia are the immune cells of the brain, but how they function in the human brain is not well understood," says Rusty Gage, professor in Salk's Laboratory of Genetics, the Vi and John Adler Chair for Research on Age-Related Neurodegenerative Disease, and a senior author of the new work. "Our work not only provides links to diseases but offers a jumping off point to better understand the basic biology of these cells."

Genes that have previously been linked to neurological diseases are turned on at higher levels in microglia compared to other brain cells, the team reported in *Science* on May 25, 2017. While the link between microglia and a number of disorders has been explored in the past, the new study offers a molecular basis for this connection.

"These studies represent the first systematic effort to molecularly decode microglia," says Christopher Glass, a Professor of Cellular and Molecular Medicine and Professor of Medicine at University of California San Diego, also senior author of the paper. "Our findings provide the foundations for understanding the underlying mechanisms that determine beneficial or pathological functions of these cells."

Microglia are a type of macrophage, white blood cells found throughout the body that can destroy pathogens or other foreign materials. They're known to be highly responsive to their surroundings and respond to changes in the brain by releasing pro-inflammatory or anti-inflammatory signals. They also prune back the connections

between neurons when cells are damaged or diseased. But microglia are notoriously hard to study. They can't be easily grown in a culture dish and quickly die outside of a living brain.

Nicole Coufal, a pediatric critical care doctor at UC San Diego, who also works in the Gage lab at Salk, wanted to make microglia from stem cells. But she realized there wasn't any way to identify whether the resulting cells were truly microglia.

"There was not a unique marker that differentiated microglia from circulating macrophages in the rest of the body," she says.

David Gosselin and Dylan Skola in the Glass lab, together with Coufal and their collaborators, set out to characterize the molecular characteristics of microglia. They worked with neurosurgeons at UC San Diego to collect brain tissue from 19 patients, all of who were having brain surgery for epilepsy, a brain tumor or a stroke. They isolated microglia from areas of tissue that were unaffected by disease, as well as from mouse brains, and then set out to study the cells. The work was made possible by a multidisciplinary collaboration between bench scientists, bioinformaticians and clinicians.

The team used a variety of molecular and biochemical tests--performed within hours of the cells being collected--to characterize which genes are turned on and off in microglia, how the DNA is marked up by regulatory molecules, and how these patterns change when the cells are cultured.

Microglia, they found, have hundreds of genes that are more highly expressed than other types of macrophages, as well as distinct patterns of gene expression compared to other types of brain cells. After the cells were cultured, however, the gene patterns of the microglia began to change. Within just six hours, more than 2,000 genes had their expression turned down by at least fourfold. The results underscore how dependent microglia are on their surroundings in the brain, and why researchers have struggled to culture them.

Next, the researchers analyzed whether any of the genes that were upregulated in microglia compared to other cells had been previously

implicated in disease. Genes linked to a variety of neurodegenerative and psychiatric diseases, they found, were highly expressed in microglia.

"A really high proportion of genes linked to multiple sclerosis, Parkinson's and schizophrenia are much more highly expressed in microglia than the rest of the brain," says Coufal. "That suggests there's some kind of link between microglia and the diseases."

For Alzheimer's, more than half of the genes known to affect a person's risk of developing the disease were expressed more highly in microglia than other brain cells.

In mice, however, many of the disease genes weren't as highly expressed in microglia. "That tells us that maybe mice aren't the best model organisms for some of these diseases," Coufal says. More work is needed to understand exactly how microglia may be altered in people with diseases, but the new molecular profile of microglia offers a way for researchers to begin trying to better culture the cells, or coax stem cells to develop into microglia for future studies.

Other researchers on the study were Baptiste Jaeger, Carolyn O'Connor, Conor Fitzpatrick, Monique Pena, and Amy Adair of the Salk Institute; Inge Holtman, Johannes Schlachetzki, Eniko Sajti, Martina Pasillas, David Gona, and Michael Levy of the University of California San Diego; and Richard Ransohoff of Biogen.

The work and the researchers involved were supported by grants from the Larry L. Hillblom Foundation, National Institutes of Health, Canadian Institute of Health Research, Multiple Sclerosis Society of Canada, University of California San Diego, Dutch MS Research Foundation, the Gemmy and Mibeth Tichelaar Foundation, the DFG, the JPB Foundation, Dolby Family Ventures, The Paul G. Allen Family Foundation, the Engman Foundation, the Ben and Wanda Hildyard Chair in Hereditary Diseases.

<http://bit.ly/2rykJhR>

Zinc may hold key to fighting liver disease

New research from Sydney's Westmead Institute for Medical Research has shown that serum zinc may benefit liver disease in an unexpected way

New research from the Westmead Institute's Storr Liver Centre in collaboration with the Centre for Virus Research and Kirby Institute has shown that serum zinc may benefit liver disease in a way we never

expected. The study, led by Dr Scott Read and Associate Professor Golo Ahlenstiel, demonstrated that zinc naturally inhibits the inflammatory and antiviral effects of interferon lambda 3 (IFN-λ3), a protein strongly associated with tissue damage in chronic liver disease. Lead author of the study, Dr Read, said the study provides the first evidence that zinc can act as a potent and specific inhibitor of IFN-λ3 in the context of viral infections such as hepatitis C and influenza.

"We have demonstrated that zinc inhibits numerous facets of the liver's immune response to viruses that may be mediated by IFN-λ3."

"Zinc interferes with IFN-λ3 binding to the interferon lambda receptor, which results in decreased antiviral activity and increased viral replication both in vitro and in vivo.

"Interestingly, zinc also blocks the inflammatory activity of IFN-λ3, which has been strongly linked to accelerated progression to liver cirrhosis in viral and non-viral liver disease. "Our data suggests that serum zinc levels in patients with chronic hepatitis C are genetically predetermined by the IFN-λ3 polymorphism, confirming the inhibitory role of zinc in vivo. "The data highlights the potential for zinc to be used as a simple and effective treatment against acute and chronic inflammation in the liver," Dr Read concluded.

Associate Professor Ahlenstiel and his team are now working towards a therapeutic intervention for IFN-λ3-mediated chronic disease. This research was published online in Nature Communications.

<http://bit.ly/2ryKkHp>

Killer antibiotic now 25,000× more potent—and resistant to drug resistance

Chemical changes give drug three killing methods plus a way to daze evolution.

[Beth Mole](#) - 5/31/2017, 11:52 PM

With clever chemical tweaks, an old antibiotic can dole out any of three lethal blows to some of the deadliest bacteria—and give evolution one nasty concussion.

The antibiotic, vancomycin, has always been a heavy hitter against odious germs; it uses one crafty maneuver that can take out even drug-resistant foes and is often used as a last resort. But, with three chemical modifications, reported this week in *PNAS*, the drug now has three distinct molecular moves to take out pathogens. The menacing modifications render vancomycin at least 25,000 times deadlier. And with that level of potency, dazed bacteria stumble at developing resistance when given the chance in lab experiments.

And maybe that should be the real goal in the war against drug-resistant microbes, the authors of the new study—chemists at The Scripps Research Institute in La Jolla, California—argue.

“As an alternative to championing the restricted use of antibiotics or conceding that bacteria will always outsmart us, can durable antibiotics be developed that are capable of continued or even more widespread use?” Perhaps, they write, we should be designing drugs that “overcome the forces of evolution and selection responsible for bacterial resistance, that are less prone or even impervious to resistance development, that avoid many of the common mechanisms of resistance, and that are more durable than ever before.”

It’s not a new idea, but it’s certainly a very hard thing to accomplish. To take a serious swing at evolution, the team, led by chemical biologist Dale Boger, built upon years of detailed structural work on vancomycin. The drug has some useful characteristics for this feat, including that bacteria naturally have trouble resisting it.

Vancomycin can kill bacteria with one of the two main types of cell wall structures—so called Gram-positive bacteria, such as *Staph aureus*. (Bacteria mainly fall into either Gram-positive or Gram-negative categories, which are based on the structures of the protective, rigid walls surrounding their cells. The structure can be figured out using [Gram staining](#), named for bacteriologist Hans Christian Gram. An example of a Gram-negative bacteria is *E. coli*.)

Knock-out drug

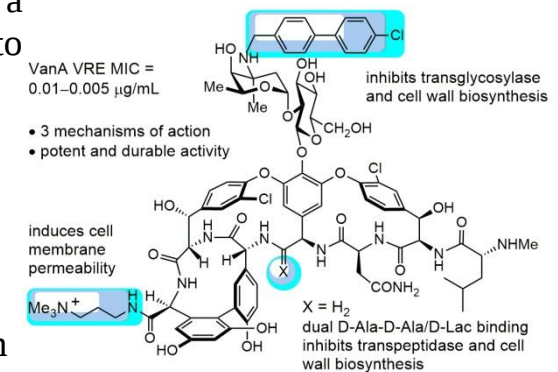
Unlike other antibiotics, which often target important enzymes or cellular machinery, vancomycin kills Gram-positive bacteria by clamping onto, basically, a molecular brick in their cell wall—linked amino acids D-Alanine-D-Alanine. Vancomycin doesn’t do anything to the brick, it just gets in the way so the wall can’t cement together properly. As such, the wall crumbles apart, destabilizing the bacteria’s structure, leading to cell death. (Gram-negative bacteria use a different wall-building method, so they’re generally safe.)

Time has shown that bacteria are bad at evolving resistance to the brick attack—there’s no simple genetic mutation to get around it. In nearly 60 years of clinical use, resistance to vancomycin has developed relatively slowly. And the resistance that has shown up is complicated and bulky: bacteria use a two-component signaling system that first senses if vancomycin is invading, then they trigger a late-stage switch in building materials, swapping D-Alanine-D-Alanine bricks for D-Alanine-D-Lactate in their walls. Facing this defense, vancomycin is a thousandfold less lethal to bacteria.

Lucky for us, there is a simple trick to defeat this cumbersome resistance: a chemical tweak to the part of vancomycin’s structure that binds to the brick can make it just as likely to glom onto D-Ala-D-Lac as D-Ala-D-Ala.

Three modifications to vancomycin's structure (shown in blue) give it new "mechanisms of action" for fighting infection. [Boger Lab](#)

With that modification, Boger and his team could crush vancomycin resistance. But they weren’t done. They also figured out how to tweak two other areas of vancomycin’s structure. On the top of the molecule, they added (4-chlorobiphenyl)methyl or CBP, which can pummel an



enzyme, called transglycosylase, involved in cell wall construction. Then, the chemists figured out that if they added a quaternary ammonium salt to the left side of the structure, this could punch holes in the cell's membrane, the delicate barrier underneath the protective wall.

Each of the three tweaks could kill bacteria on their own. But together, they make one killer antibiotic. In tests, the triple threat proved to be between 25,000 and 50,000 times deadlier to vancomycin-resistant bacteria than basic vancomycin. It also further slowed the bacteria's ability to develop resistance.

To show this, the chemists forced the hand of evolution. In labs, to goad bacteria into developing resistance to an antibiotic, all one has to do is grow generation after generation of the germs amid sub-lethal doses of the antibiotic. Boger and his team did this with their triple-threat vancomycin as well as versions of the drug that had just one or two lethal modifications. Then, they quantified how much more of each antibiotic they'd need to kill the adapted bacteria compared with the starting bacteria—a proxy for how easily the bacteria developed resistance.

After 50 growth cycles, it took up to 128-fold more of the vancomycin with one modification to kill the adapted bacteria. But for the triple-threat vancomycin, the minimum dose necessary to kill only increased four-fold.

“Such antibiotics are expected to display durable antimicrobial activity not prone to rapidly acquired clinical resistance” the authors conclude.

But before this can be put to the test in clinics, researchers will have to do animal testing and clinical trials to ensure safety and efficacy. Early toxicology work suggests that the beefed-up vancomycin is safe, however. The chemists are also working to streamline the chemical modification process, which currently requires about 30 steps.

PNAS, 2017. DOI: [10.1073/pnas.1704125114](https://doi.org/10.1073/pnas.1704125114) ([About DOIs](#)).

<http://bit.ly/2rFOFL9>

Women 'damned either way' on maternity leave

Women are judged negatively if they choose to take maternity leave - and if they don't - new research suggests.

In a study of workers' attitudes, mothers who took time off to care for babies were seen as less committed and competent at work. Meanwhile, those who continued working were viewed as less caring parents.

The results suggest women are "damned" either way, according to lead author Dr Thekla Morgenroth, of the University of Exeter.

"This is a no-win situation for women," Dr Morgenroth said.

"Our results show that perceptions of competence, whether in the work or family domain, were never boosted - but only impaired - by the maternity leave decision. "Both decisions had negative consequences, albeit in different domains.

"It is important to have policies which allow women to balance work and family life, but it's also important to understand people's use of these policies may have unintended consequences."

The study examined the attitudes of 137 women and 157 men, all employed, mostly from the US and the UK. Three groups of participants were given information about a fictional woman.

The only difference between the information was whether the woman had chosen to take maternity leave.

In one version she had taken leave, in another she had continued working, and in a third (control group) the issue was not mentioned.

Participants were then asked to evaluate the woman as a worker and a parent - with negative family results for a woman who kept working, and negative working results for a woman who took maternity leave.

"These effects occurred regardless of the respondent's gender, age, parental status or nationality - which suggests these attitudes are universal and pervasive in our culture," said Dr Morgenroth, who worked on the research along with Professor Madeline Heilman of New York University.

The majority of participants were working full-time (70%) and had no children (71%). The average age of participants was 33.32 years.

The study, published in the Journal of Experimental Social Psychology, is entitled: "Should I stay or should I go? Implications of maternity leave choice for perceptions of working mothers."

More information: Thekla Morgenroth et al, Should I stay or should I go? Implications of maternity leave choice for perceptions of working mothers, Journal of Experimental Social Psychology (2017). DOI: 10.1016/j.jesp.2017.04.008

<http://bit.ly/2sslRn6>

Rover findings indicate stratified lake on ancient Mars

Water carried more oxygen at certain times, depths

Los Alamos, N.M. - A long-lasting lake on ancient Mars provided stable environmental conditions that differed significantly from one part of the lake to another, according to a comprehensive look at findings from the first three-and-a-half years of NASA's Curiosity rover mission. While previous work had revealed the presence of a lake more than three billion years ago in Mars' Gale Crater, this study defines the lake's chemical conditions and uses Curiosity's powerful payload to determine that the lake was stratified.

Stratified bodies of water exhibit sharp chemical or physical differences between deep water and shallow water. In Gale's lake, the shallow water was richer in oxidants than deeper water was.

"We're learning that in parts of the lake and at certain times, the water carried more oxygen," said Roger Wiens, a planetary scientist at Los Alamos National Laboratory and co-author of the study, published today in the journal Science. "This matters because it affects what minerals are deposited in the sediments, and also because oxygen is important for life. But we have to remember that at the time of Gale Lake, life on our planet had not yet adapted to using oxygen--photosynthesis had not yet been invented. Instead, the oxidation state of certain elements like manganese or iron may have been more important for life, if it ever existed on Mars. These oxidation states would be controlled by the dissolved oxygen content of the water."

"These were very different, co-existing environments in the same lake," said Joel Hurowitz of Stony Brook University, lead author of the report. "This type of oxidant stratification is a common feature of lakes on Earth, and now we've found it on Mars. The diversity of environments in this Martian lake would have provided multiple opportunities for different types of microbes to survive."

Whether Mars has ever hosted any life is still unknown, but seeking signs of life on any planet, whether Earth, Mars or more-distant icy worlds, begins with reconstruction of the environment to determine if it was capable of supporting life. NASA is using Curiosity to explore habitable environments on the ancient surface of Mars.

Over more than 1,700 sols (martian days, which are 24 hours, 39 minutes long), Curiosity has traveled more than 16 km from the bottom of Gale crater part way up Mount Sharp near the center of the crater. Los Alamos National Laboratory developed the laser-shooting Chemistry and Camera (ChemCam) instrument that sits atop Curiosity in conjunction with the French space agency. Los Alamos' work on discovery-driven instruments like ChemCam stems from the Laboratory's experience building and operating more than 500 spacecraft instruments for national security. Scientists are using all the data collected by ChemCam and other on-board instruments to put together a more complete picture of the geological history of Mars.

<http://nyti.ms/2qN80Xy>

You Look Familiar. Now Scientists Know Why.

Scientists have deciphered the code of how faces are recognized

By [NICHOLAS WADE](#) JUNE 1, 2017

The brain has an amazing capacity for recognizing faces. It can identify a face in a few thousandths of a second, form a first impression of its owner and retain the memory for decades.

Central to these abilities is a longstanding puzzle: how the image of a face is encoded by the brain. Two Caltech biologists, Le Chang and Doris Y. Tsao, [reported](#) in Thursday's issue of Cell that they have deciphered the code of how faces are recognized.

Their experiments were based on electrical recordings from face cells, the name given to neurons that respond with a burst of electric signals when an image of a face is presented to the retina.

By noting how face cells in macaque monkeys responded to manipulated photos of some 2,000 human faces, the Caltech team figured out exactly what aspects of the faces triggered the cells and how the features of the face were being encoded. The monkey face recognition system seems to be very similar to that of humans.

Actual
facePredicted
faceActual
facePredicted
face

Researchers at CalTech were able to predict the appearance of faces shown to macaque monkeys simply by monitoring signals in their brains.

Doris Tsao/CalTech

Just 200 face cells are required to identify a face, the biologists say. After discovering how its features are encoded, the biologists were able to reconstruct the faces a monkey was looking at just by monitoring the pattern in which its face cells were firing.

The finding needs to be confirmed in other laboratories. But, if correct, it could help understand how the brain encodes all seen objects, as well as suggesting new approaches to artificial vision.

“Cracking the code for faces would definitely be a big deal,” said Brad Duchaine, an expert on face recognition at Dartmouth.

It is a remarkable advance to have identified the dimensions used by the primate brain to decode faces, he added — and impressive that the researchers were able to reconstruct from neural signals the face a monkey is looking at.

Human and monkey brains have evolved dedicated systems for recognizing faces, presumably because, as social animals, survival depends on identifying members of one’s own social group and distinguishing them from strangers.

In both species, the face recognition system consists of face cells that are grouped into patches of at least 10,000 each. There are six of these patches on each side of the brain, situated on the cortex, or surface, just behind the ear.

When the image of a face hits the retina of the eye, it is converted into electric signals. These pass through five or six sets of neurons and are processed at each stage before they reach the face cells. As a result, these cells receive high-level information about the shape and features of a face.

One way in which the brain might identify faces is simply to dedicate a cell to each face. Indeed, there are cells in another part of the brain that do respond to images of specific people.

They are known to neuroscientists as [Jennifer Aniston cells](#), after one such cell in an [epilepsy](#) patient undergoing surgery in 2005 responded when the patient was shown images of the actress. The cell ignored all other images, including one of her with Brad Pitt.

But this can’t be the way the brain identifies faces, because we can perceive a face we have never seen before. Instead, the Caltech team has found, the brain’s face cells respond to the dimensions and features of a face in an elegantly simple, though abstract, way.

In their experiments, the biologists first identified groups of face cells in a macaque monkey’s brain by [magnetic resonance imaging](#), and

then probed individual face cells with a fine electrode that records their signals.

The monkeys were shown photos of human faces that were systematically manipulated to show differences in the size and appearance of facial features.

Cells at a high level in the brain often respond to a medley of things, making it hard to figure out what the cell is meant to do. The Caltech team was able to create faces that showed exactly what each face cell was tuned to.

The tuning of each face cell is to a combination of facial dimensions, a holistic system that explains why when someone shaves off his mustache, his friends may not notice for a while. Some 50 such dimensions are required to identify a face, the Caltech team reports.

These dimensions create a mental “face space” in which an infinite number of faces can be recognized. There is probably an average face, or something like it, at the origin, and the brain measures the deviation from this base.

A newly encountered face might lie five units away from the average face in one dimension, seven units in another, and so forth. Each face cell reads the combined vector of about six of these dimensions. The signals from 200 face cells altogether serve to uniquely identify a face. Dr. Tsao said she was particularly impressed to find she could design a whole series of faces that a given face cell would not respond to, because they lacked its preferred combination of dimensions. This ruled out a possible alternative method of face identification: that the face cells were comparing incoming images with a set of standard reference faces and looking for differences.

Nancy Kanwisher, a neuroscientist at M.I.T., said it was a major advance to describe what a face cell does and predict how it will respond to a new stimulus. But she suggested that more than 50 dimensions might be needed to capture the full richness of human perception and the idiosyncrasies of particular faces.

“Do we need a dimension for Jack Nicholson’s eyebrows?” she asked.

Dr. Tsao has been working on face cells for 15 years and views her new report, with Dr. Chang, as “the capstone of all these efforts.” She said she hoped her new finding will restore a sense of optimism to neuroscience.

Advances in machine learning have been made by training a computerized mimic of a neural network on a given task. Though the networks are successful, they are also a black box because it is hard to reconstruct how they achieve their result.

“This has given neuroscience a sense of pessimism that the brain is similarly a black box,” she said. “Our paper provides a counterexample. We’re recording from neurons at the highest stage of the visual system and can see that there’s no black box. My bet is that that will be true throughout the brain.”

<http://bit.ly/2rpTUOh>

Timing meals later at night can cause weight gain and impair fat metabolism

Findings provide first experimental evidence of prolonged delayed eating versus daytime eating, showing that delayed eating can also raise insulin, fasting glucose, cholesterol, and triglyceride levels

BOSTON - New findings suggest eating late at night could be more dangerous than you think. Compared to eating earlier in the day, prolonged delayed eating can increase weight, insulin and cholesterol levels, and negatively affect fat metabolism, and hormonal markers implicated in heart disease, diabetes and other health problems, according to results from researchers at the Perelman School of Medicine at the University of Pennsylvania.

The findings (abstract #0064) offer the first experimental evidence on the metabolic consequences of consistent delayed eating compared to daytime eating, and will be presented at SLEEP 2017, the 31st Annual Meeting of the Associated Professional Sleep Societies LLC (APSS), on Sunday, June 4, as an oral presentation at 1:30-1:45 p.m. in room 210 and as a poster presentation from 5 p.m. to 7 p.m.

"We know from our sleep loss studies that when you're sleep deprived, it negatively affects weight and metabolism in part due to late-night eating, but now these early findings, which control for sleep, give a more comprehensive picture of the benefits of eating earlier in the day," said Namni Goel, PhD, a research associate professor of psychology in Psychiatry in the division of Sleep and Chronobiology, and lead author of the ongoing study. "Eating later can promote a negative profile of weight, energy, and hormone markers--such as higher glucose and insulin, which are implicated in diabetes, and cholesterol and triglycerides, which are linked with cardiovascular problems and other health conditions."

In the study, nine healthy weight adults underwent two conditions, one of daytime eating (i.e., three meals and two snacks between 8 a.m. and 7 p.m.) for eight weeks and another of delayed eating (i.e., three meals and two snacks eating from noon to 11 p.m.) for eight weeks. There was a two-week washout period between conditions to make sure there was no carry over effect. The sleep period was held constant, between 11 p.m. to 9 a.m.

Participants visited Penn's Center for Human Phenomic Science to get metabolic measures and blood drawn at the beginning, after the first eating condition, after the two-week washout, and after the second eating condition. This allowed the team to measure changes in weight, metabolism and energy used, and made sure the two week washout allowed all measures to return to baseline before the next condition.

The team found that when participants ate later, compared to the daytime condition, weight increased. Respiratory quotient, i.e. the ratio of carbon dioxide produced by the body to oxygen consumed by the body that indicates which macronutrients are being metabolized, also rose during the delayed eating condition, indicating later eating led to metabolizing fewer lipids and more carbs. The researchers also found that a series of other measures reflecting negative metabolic profiles increased in the delayed condition, including insulin, fasting glucose, cholesterol, and triglyceride levels.

Conducting a 24-hour hormonal profile, they also found that in during daytime eating condition, the hormone ghrelin, which stimulates appetite, peaked earlier in the daytime, while leptin, which keeps you satiated, peaked later, suggesting that the participants received cues to eat earlier, and eating earlier likely helped them to stay satiated longer. This suggests that eating earlier may help prevent overeating in the evening and at night. As sleep-wake cycles were constant, melatonin levels remained constant in both groups.

"While lifestyle change is never easy, these findings suggest that eating earlier in the day may be worth the effort to help prevent these detrimental chronic health effects," said Kelly Allison, PhD, an associate professor of psychology in Psychiatry and director of the Center for Weight and Eating Disorders, and senior author on the study. "We have an extensive knowledge of how overeating affects health and body weight, but now we have a better understanding of how our body processes foods at different times of day over a long period of time."

Similar yet much shorter previous studies have suggested similar results, but this is the first long-term study looking at the timing of eating patterns that also controlled for sleep-wake cycles, exercise, macronutrient intake, etc. to pinpoint the effects of prolonged eating at different times of day.

This study was funded by the National Institutes of Health (R21 DK100787). Additional authors on the study include Christina Hopkins, Madelyn Ruggieri, Andrea Spaeth and Rexford Ahima, all from Penn.

<http://bit.ly/2rGbH4p>

Life on terra firma began with an invasion

Scientists are now confident animal life on solid ground started with a few short bursts of marine creatures making the leap from the oceans.

New research at the University of Portsmouth also paints a clear picture of how animals rapidly spread out and changed once they made the leap. The research, led by Dr Nicholas Minter of the

University's School of Earth and Environmental Sciences, is published in *Nature Ecology and Evolution*.

The first study to use trace fossils – the footprints of animal behaviour such as tracks and burrows – shows how a major evolutionary step for the Earth, the colonisation of land, took place. The initial invasion happened across the world, from the fringes between land and oceans, followed by expansion later into floodplains, rivers, deserts and lakes. It is the first time such a pattern has been identified using trace fossils to track the evolution of animal behaviour, their modes of life, and the ways in which they interacted with their new environments.

Dr Minter said: "When the first animals emerged from the oceans they had a blank canvas, there were no other animals there and so they diversified rapidly both in how they behaved and in the ecological roles, or the part they played, in the theatre of life."

This evolutionary step-change – an early burst of rapidly adapting and diversifying to new environments, followed by a long period of little change – has been seen in the shapes and sizes of animals but this is the first time it has been shown for their behaviour.

The creatures which made the leap to land represent just a handful of the marine population, with spiders coming from the same group as crabs, and slugs and snails coming from the same group as squid and cuttlefish, for example.

"Not many from any one group, or Phylum, made the transition from sea to land or fresh water," said Dr Minter.

"What's surprising is the leaps in evolution follow the same pattern – an early evolutionary burst of rapid diversification and a long period of relative calm – each time animals conquered new habitats, first the margins between sea and land, then floodplains, followed by rivers, deserts and lakes. "Each burst was an evolutionary experiment, yet the results are very similar.

"As scientists, we know experiments tend to have different outcomes if you change the parameters, but in this case, there is a consistency

which suggests a fundamental constraint on the behavioural and ecological roles fulfilled by animals on land."

The exhaustive study – a seven-year-long analysis of all known trace fossil data spanning a 200 million year period of Earth history – has the potential to tell us about our future, too, he said.

"We now have a framework we can use to apply to other questions and allows us to compare ancient and living communities. This framework may help us understand if modern communities are undergoing fundamental changes, as ancient ones have in the past, for example, in response to climate change."

Trace fossils tell us a great deal about animal life, including who was there, when and how they behaved. Although Dr Minter's research included examining reports of hundreds of trace fossils from hundreds of rock formations around the globe, the planet has not given up all its secrets.

He said: "This is not necessarily the final word. Another major step-change that took place was the evolution of social insects, such as ants and termites, which worked together to build complex structures and societies. More recently too, in the evolution of animals was the emergence of intelligent life – animals which could make and use tools, including humans and our ancestors, apes. We are yet to investigate how animals diversified and modified their environments during these events and how this shaped our planet."

<http://nyti.ms/2rG6eL4>

The Mummies' Medical Secrets? They're Perfectly Preserved

Mummified bodies in a crypt in Lithuania are teaching scientists about health and disease among people who lived long ago.

By [NICHOLAS ST. FLEUR](#) JUNE 2, 2017

Hundreds of skeletons have lain scattered around a crypt beneath a church in Vilnius, Lithuania, for centuries. But 23 of these remains are unlike the rest: Flesh wraps their bones, clothes cover their skin, and organs still fill their insides.

They are mummies, and since they were recovered about five years ago, scientists have investigated their secrets, seeking insights into the lives of people in the 17th, 18th and 19th centuries and the diseases they suffered.

“They are so well preserved that they almost look alive,” said [Dario Piombino-Mascali](#), an anthropologist from Italy who has studied the mummies since 2011.

Recently, Dr. Piombino-Mascali and his colleagues have uncovered remnants of the smallpox virus in one of the mummies, gaining new insights into the origins of a deadly scourge that killed an estimated 300 million people in the 20th century alone.



One of 23 mummies found in a crypt under a church in Vilnius, Lithuania.

Kiril Cachovski of the Lithuanian Mummy Project, 2015

The work follows on their earlier discoveries: signs of rickets, osteoarthritis and intestinal parasites in the mummies. And they are not the only researchers unearthing new findings from the bodies of the long dead but well preserved.

The study of mummified remains in other parts of the world has yielded historical perspective on the spread of deadly diseases and damaging medical conditions, from heart disease in pre-Columbian Americans to various strains of tuberculosis in 19th-century Europeans.

By understanding how long these diseases have been around and mapping them historically, scientists can better tackle them today.

“Most people don’t realize you can learn about modern medicine from ancient mummies,” said [Dr. Frank Rühli](#), head of the [Swiss Mummy Project](#) at the University of Zurich in Switzerland, who is studying the internal organs of Iranian and Egyptian mummies.

“These historic patient records are like a box of candy for us.”

A crypt in the heart of a capital

In the heart of Vilnius, Lithuania’s Dominican Church of the Holy Spirit is a bright masterpiece of Late Baroque architecture. But it hides something darker.

Inside, an altar stands behind a large wooden platform where people kneel and pray. Beneath this is a stone staircase so narrow it can admit only one person at a time. Researchers liken it to an entrance to a secret lair: The steps descend to a dark and dusty underworld.

A black metal gate leads to the labyrinthine chambers that house the corpses. Once, there were body parts piled into a pyramid on the floor and stacked on shelves that reached to the ceiling.

For most of their history, the corpses were preserved intact: Cool temperatures and ventilation in the underground chamber had caused them to undergo spontaneous mummification. ([Read more about how bodies spontaneously mummify.](#)) Though they faced disturbances over the centuries as the city and church were occupied by Napoleon, then the Nazis, it was the Soviet occupation of Lithuania that brought about a drastic change in the mummies’ fate.

In the 1960s, a forensic scientist named [Juozas Albinas Markulis](#) became one of the first to study the mummies. He wanted to know whether there were victims from World War II mixed in among the 17th-, 18th- and 19th-century corpses. (Oddly, Dr. Markulis is better known to Lithuanians not as a scientist but as a former spy who, while posing as a leader of the Lithuanian resistance, lured others into Soviet ambushes.)

Dr. Markulis and his students at Vilnius University identified 500 bodies in the crypt, of which about 200 had been mummified. In 1962, government officials inspected the crypt and ordered that the mummies be sealed behind glass, fearful that infected bodies might start an epidemic. They called it the Chamber of Death.

Soon a glass wall was erected, but it stopped the airflow and made the environment too humid and caused the mummies to decay. Dr. Markulis tried to save them, but his pleas were ignored by the

Lithuanian government. The site was soon closed and remained unstudied until anthropologists returned to the chamber in 2004.

From 2008 to 2011, researchers began inspecting and extracting the mummies from the crypt. Of the 200 studied by Dr. Markulis, who died in 1987, only 23 remained intact.

But while Dr. Markulis sought to uncover the identities of the mummies, Dr. Piombino-Mascali and his colleagues focused on how they lived.

Mummified medical mysteries

Looking at the remains, Dr. Piombino-Mascali identified several with dental decay and gum disease, as well as arthritis and bone deformities. To further investigate their health issues, he performed C.T. scans on the seven best-preserved mummies.

One obese man once had arthritis in his spine, pelvis and both knees, a fractured rib on his right side and an enlarged thyroid gland, which might have been caused by goiter. An obese woman had a benign tumor in her lower back. Both had suffered from clogged arteries, a health problem usually associated with modern diets.

“It was very strange,” Dr. Piombino-Mascali said of the examinations, “because we were not feeling as if they were just objects of cultural and archaeological interest. It was a feeling like they were with us willing to do a checkup for their medical conditions.”

The researchers sent samples from a 17th century mummified child to a colleague in Canada, who uncovered [remnants of variola virus](#) that causes smallpox, which once ravaged most of the world. By sequencing the virus, the team has gained insight into the origins of the deadly scourge.

“There was no evidence on any remains that would suggest a smallpox infection, so the presence of variola virus was very surprising,” said [Ana Duggan](#), a biologist from McMaster University who worked with Dr. Piombino-Mascali. “It’s the oldest complete genome that we have of variola virus.”

She said the ancient DNA has helped them map out the timeline of smallpox. Historical accounts from Egypt, China and India had suggested that smallpox had infected humans for thousands of years. But by comparing the 17th century strain with modern variola samples, they found the strains shared a common ancestor that emerged between 1530 and 1654. Their finding suggests that the deadliest kinds of smallpox may have evolved much more recently than previously thought.

The discovery in the Lithuanian crypt is one of the latest in a long line of important medical findings that have used intensive analysis of mummified to show how diseases connect modern humans to the experiences of our forebears.

In 2013 a team led by [Dr. Randall C. Thompson](#), a cardiologist at St. Luke’s Mid America Heart Institute in Kansas City, Mo., performed [C.T. scans on 130 mummies](#) from ancient Egypt and pre-Columbian Peru, as well as those of Native Americans in the Southwest and the Unangan people of the Aleutian Islands.

He and his colleagues discovered that more than a third of the mummies had some form of atherosclerosis, or hardening of the arteries, which can lead to heart disease. The affected mummies came from various geographical regions and lived over a span of more than 4,000 years — a reminder that heart troubles have long been prevalent and are not simply the result of modern diets.

“We found that heart disease is older than Moses,” he said. “This disease was present and not hard to find all over the world covering a wide swath of human history.”

Using burial markings on the tombs, they identified their oldest case of coronary heart disease in Ahmose-Meritamun, an Egyptian princess who lived from 1550 to 1580 B.C. and was in her 40s when she died. The oldest example of clogged arteries they found belonged to an Egyptian mummy from around 2,000 B.C.

[Dr. Mark Pallen](#), a professor of microbial genomics at the University of Warwick, made similar findings in 2015 while studying

[tuberculosis in mummies](#) found in a Hungarian crypt with more than 200 bodies.

He and his team had extracted tuberculosis bacteria DNA from the lungs of eight 200-year-old mummies, discovering that ancient people could get multiple strains of the bacteria throughout their lifetimes.

They used a technique known as metagenomics sequencing, which had not been previously used on mummified corpses. It allowed the researchers to extract microbial DNA directly rather than having to grow the bacteria on a plate.

Because of its success in mummies, he had used the technique with mucus samples from people to get [DNA from tuberculosis bacteria](#).

"In this case, the dead did instruct the living," Dr. Pallen said.

Back in Lithuania, Dr. Piombino-Mascali said cases of both atherosclerosis and tuberculosis had been found among mummies in the church crypt. The findings offered evidence that even the upper class in 18th- and 19th-century Vilnius experienced chronic health problems, including those related to poor nutrition.

But most important to Dr. Piombino-Mascali, the mummies now are sharing their stories.

"That crypt was a witness to all of the historical faces of Vilnius," Dr. Piombino-Mascali said. "But now it's finally given back to the city. The stories belong to Lithuania and especially the Lithuanian people."

<http://bit.ly/2s6mZ2I>

Adding abiraterone to standard treatment improves prostate cancer survival by 40 percent

Adding abiraterone to hormone therapy at the start of treatment for prostate cancer improves survival by 37 per cent

Adding abiraterone to hormone therapy at the start of treatment for prostate cancer improves survival by 37 per cent, according to the results of one of the largest ever clinical trials for prostate cancer* presented at the 2017 ASCO Annual Meeting in Chicago and published in the New England Journal of Medicine today (Saturday).

The results from the Cancer Research UK-funded STAMPEDE** trial could change the standard of care for men with prostate cancer, making abiraterone a first-line treatment alongside hormone therapy.

This part of the STAMPEDE trial recruited around 1,900 patients.***

Half the men were treated with hormone therapy while the other half received hormone therapy and abiraterone. In men who were given abiraterone there was a 70 per cent reduction in disease progression.****

The drug is usually given to men with advanced prostate cancer that has spread and has stopped responding to standard to hormone therapy, but this study shows the added benefit to patients who are about to start long-term hormone therapy.

Professor Nicholas James, chief investigator of the Cancer Research UK-funded STAMPEDE trial from the University of Birmingham, said: "These are the most powerful results I've seen from a prostate cancer trial -- it's a once in a career feeling. This is one of the biggest reductions in death I've seen in any clinical trial for adult cancers.

"Abiraterone is already used to treat some men whose disease has spread but our results show many more could benefit. In addition to the improvements in survival and time without relapse, the drug reduced the rates of severe bone complications, a major problem in prostate cancer, by more than a half. I really hope these results can change clinical practice."

Alfred Samuels, 59, was diagnosed with advanced prostate cancer in January 2012 and joined the STAMPEDE trial two months later. He lives in Harrow with his partner and five children.

Alfred said: "It felt like my world fell apart overnight. The doctors explained that surgery wasn't an option for me because the cancer had spread beyond my prostate. "But my clinician suggested that the STAMPEDE trial might be a good option. As part of the trial, I started taking abiraterone four times a day and had a hormone injection every eight weeks. "During the first six months, tests showed that the treatment was working. I'm still on the trial, which I find reassuring and, fortunately, my cancer is being managed well."

Prostate cancer cells usually depend on testosterone to grow. Standard hormone therapy blocks the action of male sex hormones, halting the disease. Abiraterone goes further and shuts down the production of the hormones that fuel prostate cancer's growth.

Each year around 46,500 men are diagnosed with prostate cancer in the UK, and around 11,000 men die from the disease.

Sir Harpal Kumar, Cancer Research UK's chief executive, said: "These results could transform the treatment of prostate cancer.

Abiraterone can clearly help many more prostate cancer patients than was first thought.

"The STAMPEDE trial is changing the face of prostate cancer because the flexibility of the trial design means that we can investigate a number of different treatment options rapidly and in parallel, enabling scientists to get results much more quickly than they usually would. "Cancer Research UK scientists first discovered abiraterone and subsequently played a key role in its development, including funding the first clinical trials. This study adds to the importance of the drug."

The STAMPEDE trial is coordinated by the MRC Clinical Trials Unit at UCL and funded by Cancer Research UK. Previous results from the trial have already changed clinical practice - data released last year has led to docetaxel chemotherapy now being part of the standard of care for many men with prostate cancer. More data will come out in subsequent years, because of the innovative design of the trial. Professor James added: "We are so incredibly thankful to the patients and clinical staff who have agreed to take part in this study. With their generosity, scientists can carry out research that will help save lives."

** James et al. Abiraterone Acetate Plus Prednisolone for Hormone-Naïve Prostate Cancer. New England Journal of Medicine ASCO Abstract LBA5003: Adding abiraterone for men with high-risk prostate cancer (PCa) starting long-term androgen deprivation therapy (ADT): Survival results from STAMPEDE (NCT00268476).*

*** The Cancer Research UK funded STAMPEDE trial is the largest ever prostate cancer trial with over 9,000 men.*

STAMPEDE trial: <http://www.cancerresearchuk.org/about-cancer/find-a-clinical-trial/a-trial-looking-at-hormone-therapy-with-zoledronic-acid-docetaxel-or-celecoxib-for-prostate-cancer>

STAMPEDE is a Cancer Research UK grant-funded trial which is sponsored and support by core-funding from the UK Medical Research Council; and conducted by the MRC Clinical Trials Unit at UCL. Janssen provided a further educational grant for this comparison. Further funding includes Astellas, Clovis, Novartis, Pfizer and Sanofi-Aventis.

Cancer Research Technology, the commercial arm of Cancer Research UK, receives a small royalty from the drug's sales which are reinvested in Cancer Research UK's lifesaving work.

**** 957 men in the control arm received standard care (androgen deprivation therapy) and 262 died within four in a half years, whereas 960 men received abiraterone in combination with standard care, 184 of which died.*

*****Failure free survival included no drastic change in PSA, no spread to lymph nodes or other metastasis, or death from prostate cancer.*