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Signs of Alien Air Herald a New Era of Exoplanet Discoveries

New efforts hint that nearby world GJ 1132 b may have an Earth-like atmosphere with water and methane

By Mara Johnson-Groh on January 23, 2017

For astronomers seeking Earth twins around other stars, the exoplanet GJ 1132 b probably isn't an identical sibling—but it may be the closest cousin yet found. It weighs in at just over one Earth mass, but circles its star in a warm orbit that could make it more like Venus than our own world. Moreover, its diameter is nearly 50 percent larger than that of Earth, suggesting it possesses a thick atmosphere.

Now, after taking the closest-ever look at GJ 1132 b, a European collaboration has confirmed the presence of its atmosphere and found hints it might contain water and methane. The results are currently under review for publication in The Astrophysical Journal.

As mere discoveries of exoplanets become routine, efforts to learn more about them—their compositions, climates and histories—are moving to the fore, with studies of their atmospheres occupying center stage. Although astronomers detected the first exoplanet atmosphere more than 15 years ago, they have only managed to observe a handful ever since, mostly for very hot worlds as big as Jupiter or even larger. With their first glimpse of GJ 1132 b's alien air, astronomers are now entering a new frontier as they examine the atmospheres of smaller, more Earth-like worlds.

"We have shown that an Earth-mass planet is capable of sustaining a thick atmosphere," says John Southworth, lecturer in astrophysics at Keele University in England and lead author on the discovery paper. "This is one step towards investigating whether a planet could host life."

Finding the tenuous atmospheres around other worlds strains the limits of current technology. Luckily, GJ 1132 b has the advantage of being relatively easy to study, because it is only 39 light-years away—just a hop, skip and a jump across our cosmic neighborhood. It also orbits an M-dwarf, the smallest and coolest type of star, which allows astronomers to more readily probe the planet's atmosphere.

"Detecting the atmosphere of Earth-sized planets around M-dwarfs is an essential step in the search for habitable exoplanets," says astronomer Julien de Wit, postdoctoral researcher at Massachusetts Institute of Technology unaffiliated with the study. "The concern, however, is that they may not always be able to sustain an atmosphere because of the potential history of strong activity of their star. Finding one with an atmosphere would provide us with hope."

The team studied GJ 1132 b's atmosphere using a variation on the "transit" planet-detection method, in which a world traverses the face of its star as seen from Earth. As the planet crosses its star, it blocks a small portion of the star's total light and casts a planetary shadow toward our solar system. A planet's atmosphere will absorb a tiny fraction of starlight around the shadow's edges, filtering out certain wavelengths in accordance with its composition.

Gathering enough light to discern this minuscule effect usually requires observing multiple transits using some of the world's most powerful telescopes.

Using the MPG/ESO 2.2 meter telescope at the European Southern Observatory in Chile, the team monitored nine transits of GJ 1132 b across a wide range of wavelengths, from optical to near-infrared. The measurements allowed them to line up a simple spectrum, which shows the amount of light at each wavelength.

Their results showed extra absorption at certain wavelengths—indicating the possibility of water and/or methane in GJ 1132 b's atmosphere in approximately equal proportions as is found in Earth's air.

Because astronomers have good measures for both the mass and the size of GJ 1132 b, they can estimate the planet's density—and thus its possible composition. Given that the atmosphere may contain water vapor, one model suggests the planet could be a steamy space oasis

with a substantial envelope of water surrounding a rocky core. Other models with rockier compositions are also possible, however, and the mass measurements are not detailed enough to wholly confirm the exact interior makeup. “Our own observations have shown that it has an atmosphere, but we have not been able to put many constraints on what the atmosphere is actually made of,” Southworth says. “The next step is to take observations with bigger telescopes, and space telescopes, covering a bigger wavelength range at a much better resolution.”

Given the limitations of current instruments, it will fall to the next generation of telescopes such as the James Webb Space Telescope (JWST) to glean more information about the nature of Earth-size exoplanet atmospheres.

“The James Webb Space Telescope will be able to measure the spectra of exoplanets in great detail, and perhaps GJ 1132 b will be one of the more interesting exoplanets to be observed extensively” with the JWST, says Renyu Hu, a planetary scientist at the NASA Jet Propulsion Laboratory. “We will continue to see astronomers pushing the limits, towards better spectra of smaller and perhaps more temperate exoplanets.”

Boasting a mirror some six times the size of the Hubble Space Telescope’s, JWST will be able to efficiently seek out signs of carbon dioxide and oxygen as well as water vapor and methane in some exoplanet atmospheres. Eager planet hunters, however, should not hold their breath for these breakthrough capabilities—JWST will not launch until October 2018, and the telescope has a full docket of other science objectives that will limit any time-consuming observations of exoplanets.

“This is a great start, but we need higher signal-to-noise data and spectral resolution,” Sara Seager, a professor of astrophysics at MIT, says of the results. “We need to await the JWST to make any real progress on small-planet atmospheres.”

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New research debunks honey bee pesticide study

Study by global agrochemical company described as "misleading”

A study by a global agrochemical company that concluded there was only a low risk to honey bees from a widely used agricultural pesticide has been described as "misleading" in new research published by statisticians at the University of St Andrews.

Pesticides called neonicotinoids or neonics may be implicated in losses of honey bees and other pollinators. The economic value of honey bees and bumble bees on the pollination of commercially grown crops has been estimated at over £200 million a year in the UK alone.

A major study conducted by Swiss agrochemical company Syngenta on the effects of the neonic thiamethoxam on honey bees in the field concluded that there was only a low risk to honey bees.

New research conducted at the Centre for Research into Ecological and Environmental Modelling (CREEM) by Dr Robert Schick, Professor Jeremy Greenwood and Professor Steve Buckland shows even large and important effects could have been missed because the Syngenta study was statistically too small. Their findings are published today in the international journal Environmental Sciences Europe.

The Syngenta study involved two experiments: an oilseed rape experiment conducted at two locations and a maize experiment at three locations. At each location the experiments used pairs of fields – in one field the crop was treated with thiamethoxam at levels normally used by farmers, in the other field the crop was untreated.

The Syngenta study concluded that because the experiments involved so little replication (two cases for oilseed rape and three for maize) a formal analysis of the data "would lack the power to detect anything other than very large treatment effects, and it is clear from a simple inspection of the results that no large treatment effects were present.

Therefore a formal statistical analysis was not conducted because this would be potentially misleading".

The St Andrews team believe this is fundamentally wrong because formal statistical analysis is only potentially misleading if the wrong method is used and because the mere inspection of the results is always potentially misleading because it is an entirely subjective procedure.

Professor Greenwood said: "In order to reach valid conclusions about the results of an experiment such as this, one needs not just to estimate the effect of the treatment but also to measure the precision of the estimate. That is what we have done, using standard statistical techniques.

"What we found was that the estimates of the treatment effects were so imprecise that one could not tell whether the effects were either too small to pose a problem or, in contrast, so large as to be of serious concern. "In effect, the experiments were on such a small scale that little useful could be concluded from them."

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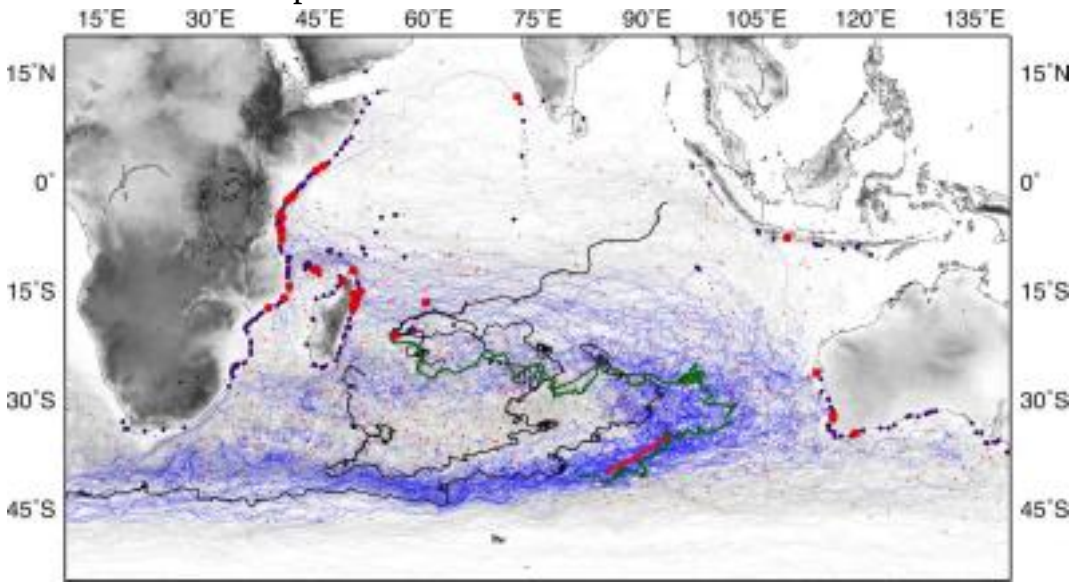
Oceanographic analysis offers potential crash site of MH370

Researchers use data from ocean drifters to aid analysis

MIAMI -- A group of oceanographers offers a new analysis of the potential crash site of flight Malaysian Airlines flight 370 in the southern Indian Ocean. The researchers, which included scientists from the University of Miami (UM) Rosenstiel School of Marine and Atmospheric Science, used data from buoys that monitor ocean conditions.

In their analysis the team considered the trajectories of drifting buoys, called drifters, from NOAA's Global Drifter Database and of an ocean numerical model. The researchers included only those data from drifters that were unanchored, or undrogued, to better simulate the buoyancy conditions of airplane debris. The team then produced a simulation model of drifter motion using known oceanographic conditions near the potential crash site.



Trajectories of undrogued surface drifters from the historical dataset. The blue trajectories correspond to drifters washing ashore (otherwise the grey colour is used). The small red dots represent the location of their latest transmission. The larger circles refer to drifters reaching land: in red, for the drifters that at some time travelled through the search area; in blue, the same but for the indirect trajectories. The thicker trajectories represent those drifters arriving at Reunion Island. The one in green represents a trajectory that at some point passed through the search area.

J. A. TRINANES ET. AL.

The analysis showed that it would take six months to one year for the drifters to reach western Australia and one-and-a half to two years to reach eastern Africa. Interestingly, two drifters traveled from the search region to the area of Reunion Island during the period between the crash of flight MH370 and when the airplane flaperon was found.

These results are consistent with the time and location of the aircraft debris that was found off Reunion Island, almost 17 months after the plane disappeared, and with the recently confirmed finding in Mozambique almost two years later.

The trajectories of the undrogued drifters and synthetic drifters revealed several areas of high probability in the southern Indian Ocean where debris from the missing flight could have passed, including vast areas of the South Indian Ocean, some of them in the relative neighborhood of the search area.

This study "highlights the importance of sustained observations to monitor ocean conditions that may serve a suite of applications and studies," said the authors. The methods developed by the researchers for use in the study could also help scientists track oil spills, and other types of marine debris and pollutants in the ocean.

The study, titled "Analysis of flight MH370 potential debris trajectories using ocean observations and numerical model results," was recently published online in the Journal of Operational Oceanography. The coauthors include: M. Josefina Olascoaga from the UM Rosenstiel School; Joaquin A. Trinanes and Gustavo J. Goni from NOAA's Atlantic Oceanographic and Meteorological Laboratory in Miami; Nikolai A. Maximenko and Jan Hafner from the University of Hawaii's School of Ocean and Earth Science and Technology; and David A. Griffin from CSIRO in Australia.

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Transplanted neurons incorporated into a stroke-injured rat brain

Transplanted neurons in the brains of stroke-injured rats were incorporated and responded correctly

Today, a stroke usually leads to permanent disability - but in the future, the stroke-injured brain could be reparable by replacing dead cells with new, healthy neurons, using transplantation. Researchers at Lund University in Sweden have taken a step in that direction by showing that some neurons transplanted into the brains of stroke-injured rats were incorporated and responded correctly when the rat's muzzle and paws were touched.

The study, published in the prestigious journal Brain, used human skin cells. These cells were re-programmed to the stem cell stage and then matured into the type of neurons normally found in the cerebral cortex. A couple of years ago, the research team at the Stem Cell Centre in Lund had already proven that transplanting this type of cells to the cerebral cortex enabled stroke-injured rats to move better. At the time, however, it was unclear whether the host brain really formed functioning connections with the transplanted nerve cells. Now the new study has proven that this is indeed the case.

The research team used several advanced methods in the study - electron microscopy, virus-based tracing techniques, registration of activity in the transplanted cells and optogenetics. The results show that various parts of the host brain form normal, functioning connections with the transplanted neurons and that the latter change their activity when the animal's muzzle and paws are touched.

"This is the first time anyone has been able to show such a result. That some of the new nerve cells receive signals from the host brain in a normal way indicates that they have been incorporated into the stroke-injured rat's brain. In it, they have been able to replace some of the dead nerve cells", says the professor at the Stem Cell Centre, Zaal Kokaia.

Now, a stroke-injured laboratory animal is not the same as a stroke patient. But professor and consultant physician Olle Lindvall, who is also part of the research team, still sees the team's study as an important first step. It constitutes what is known as proof of concept, showing that it is possible to replace dead neurons with new, healthy cells through transplantation after a stroke.

"This is basic research, and it is not possible to say when we will be ready to start experiments on patients. But the objective is clear: to develop a treatment method which can repair the stroke-injured brain. Currently, there is no effective treatment which can restore function in a stroke patient once the first hours following a stroke have passed", says Zaal Kokaia.

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How do people choose what plants to use?

From medicine to rituals

There are about 400,000 species of plants in the world. Humans use approximately 10-15% of them to cover our basic needs, such as food, medicine and shelter, as well as other needs, such as recreation, art,

and craft. But why and how have humans selected only a small fraction of all plants to utilize? A new study published in today's Nature Plants sheds new light on these questions by investigating how people use palms in South America. The overall conclusion is that people are very selective when it comes to plants used to cover basic needs, but less so when it comes to using plants for needs with no physiological underpinnings.

We know that certain plant traits, such as taste and scent, can affect how we perceive plants. For example, if a fruit tastes sweet, it is very likely that we will eat it. If the leaves of a plant have a mint-like scent, it is likely that we will use these leaves as herbs or tea.

However, plants come in all shapes and sizes and possess several traits that affect how we think of them, and until now it has been unclear exactly how plant traits determine how we use plants to cover our needs.

Large, widespread species

In this study, we investigated how people use palms in South America. We focused on palms because they are very important for local livelihoods in several parts of the world, including South America. We interviewed 2,200 locals from over 60 communities about how they use palms and we collected data on biological traits of these palms, such as plant size (leaves, fruits, stems) and range size. We then tested for the correlation of these traits with the perceived value of the different palm species, says Rodrigo Cámara-Leret from the Royal Botanic Gardens, Kew, and continues

We found that people tend to use large, widespread species compared to small, narrow-ranged ones. For example, people prefer larger palms for food, potentially because they need palms that produce large quantities of food, rather than ones that are small and produce less

Species for basic needs

More importantly, we found that the more basic the human need that a plant covers, the stronger the link with biological traits. Palms used for basic physiological and safety needs (e.g. food, medicine, shelter) have a very strong link to plant size (the bigger, the better) and range size (the more the merrier).

On the other hand, palms used for psychological and self-actualization needs (e.g. rituals, jewelry) are less dependent on the biological traits of palms. In other words, people are very selective when it comes to plants used to cover basic needs, but less so when it comes to using plants for needs with no physiological underpinnings.

Overlooked species

These findings can also have implications about the ways we can utilize wild plants in order to meet societal needs in a changing world with a growing human population.

Our results suggest that reliance on plant size and availability may have prevented our optimal exploration of the properties of wild plants, since small and rare plants might have been overlooked. These overlooked species might present an untapped natural resource that merits further investigation, says Haris Saslis-Lagoudakis, Assistant Professor at the Natural History Museum of Denmark.

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World still 'grossly underprepared' for infectious disease outbreaks

The world remains "grossly underprepared" for outbreaks of infectious disease, which are likely to become more frequent in the coming decades, warn a team of international experts in The BMJ today.

They reviewed reports on the recent Ebola virus outbreak in West Africa and say better preparedness and a faster, more coordinated response could have prevented most of the 11,000 deaths directly attributed to Ebola and also the broader economic, social, and health crises that ensued.

In August 2014, the World Health Organization (WHO) declared the Ebola outbreak in West Africa a Public Health Emergency of International Concern (PHEIC), and the world scrambled to respond.

In the aftermath, a number of reports were published reviewing what went wrong and how we should better manage infectious disease outbreaks. However, the main priorities emerging from these reports and the extent to which action has been taken on the proposed reforms is unclear.

So a research team, led by Suerie Moon at the Graduate Institute of International and Development Studies in Geneva, synthesized seven major post-Ebola reports and laid out the key problems and recommendations they highlighted.

They also assessed progress to date and identified the biggest gaps between recommendations and action in each area of reform.

They found that, while the reports differed in scope and emphasis, their diagnosis of the key problems and recommendations for action converged in three critical areas: strengthening compliance with the International Health Regulations (IHR); improving outbreak-related research and knowledge sharing; and reforming the World Health Organization (WHO) and broader humanitarian response system.

They found significant efforts beginning to address these issues, but that progress has been mixed with many critical issues largely unaddressed.

For example, they point out that investments in country capacity building have been inadequate and difficult to track, arrangements for fair and timely sharing of patient samples remain weak, and reform efforts at WHO have focused on operational issues but have neglected to address deeper institutional shortcomings.

As the WHO Executive Board gathers this week to shortlist candidates in the running for the 2017 WHO Director-General election, the authors point out that "spearheading institutional reforms is likely to fall to the next director general."

"We found remarkable consensus on what went wrong with the Ebola response and what we need to do to address the deficiencies. Yet not nearly enough has been done," write the authors.

"Ebola, and more recently Zika and yellow fever, have demonstrated that we do not yet have a reliable or robust global system for preventing, detecting, and responding to disease outbreaks," they add.

And they urge the global community "to mobilize greater resources and put in place monitoring and accountability mechanisms to ensure we are better prepared for the next pandemic."

"We will not be ready for the next outbreak without deeper and more comprehensive change," they conclude.

Analysis: Post-Ebola reforms: ample nnalysis, inadequate action http://www.bmj.com/cgi/doi/10.1136/bmj.j280

http://bit.ly/2kmuIlp

ASU scientist finds advanced geometry no secret to prehistoric architects in US Southwest

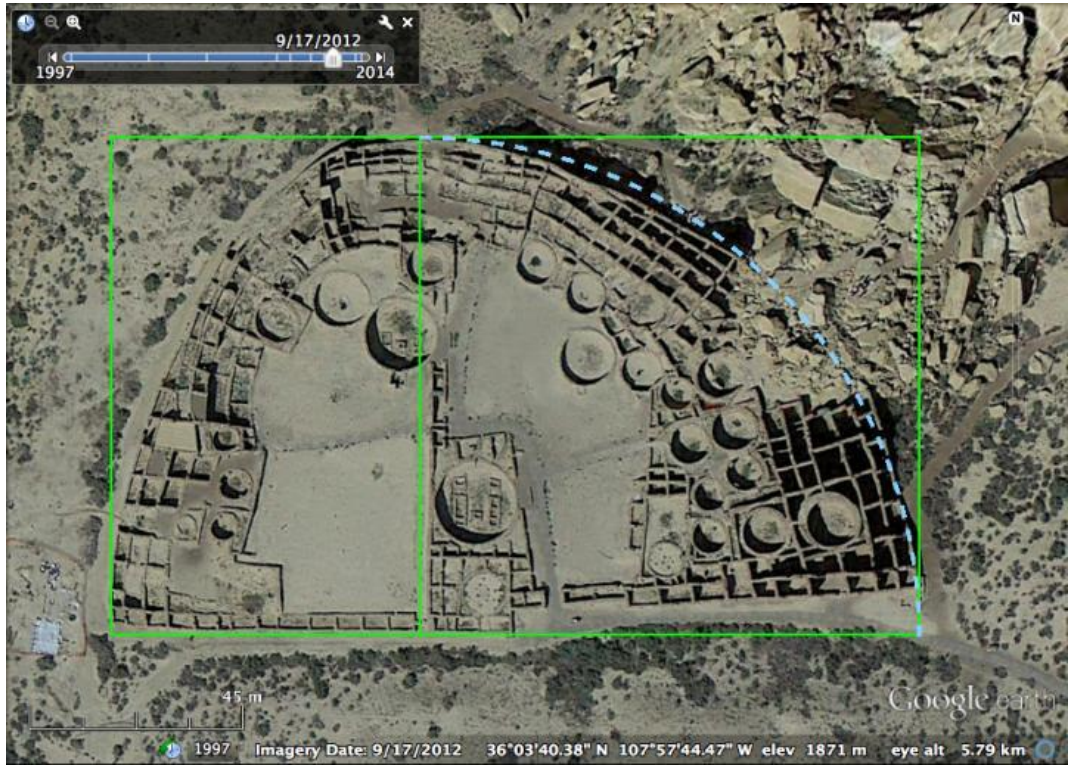
Ancient Southwestern Pueblo people with no written language or written number system were able to plan and construct complex buildings

Imagine you are about to plan and construct a building that involves several complicated geometrical shapes, but you aren't allowed to write down any numbers or notes as you do it. For most of us, this would be impossible.

Yet, new research from Arizona State University has revealed that the ancient Southwestern Pueblo people, who had no written language or written number system, were able to do just that - and used these skills to build sophisticated architectural complexes.

Dr. Sherry Towers, a professor with the ASU Simon A. Levin Mathematical, Computational and Modeling Sciences Center, uncovered these findings while spending several years studying the Sun Temple archaeological site in Mesa Verde National Park in Colorado, constructed around A.D. 1200.

"The site is known to have been an important focus of ceremony in the region for the ancestral Pueblo peoples, including solstice observations," Towers says. "My original interest in the site involved looking at whether it was used for observing stars as well."



This is a satellite photo of Pueblo Bonito archaeological site with illustrations demonstrating its geometrical properties. **Dr. Sherry Towers**

However, as Towers delved deeper into the site's layout and architecture, interesting patterns began to emerge.

"I noticed in my site survey that the same measurements kept popping up over and over again," she says. "When I saw that the layout of the site's key features also involved many geometrical shapes, I decided to take a closer look."

The geometrical shapes used within this location would be familiar to any high school student: equilateral triangles, squares, 45-degree right triangles, Pythagorean triangles, and the "Golden rectangle," which was well known to architects in ancient Greece and Egypt and is often used in Western art due to its pleasing proportions.

With some geometrical know-how, a straight-edge, a compass or cord, and a unit of measurement, all of the shapes are fairly easy to construct. But, unlike the ancient Greeks, Egyptians and Maya, the ancestral Pueblo people had no written language or number system to aid them when they built the site. Incredibly, their measurements were still near-perfect, with a relative error of less than one percent.

"This is what I find especially amazing," Towers says. "The genius of the site's architects cannot be underestimated. If you asked someone today to try to reconstruct this site and achieve the same precision that they had using just a stick and a piece of cord, it's highly unlikely they'd be able to do it, especially if they couldn't write anything down as they were working."

During her research, Towers discovered that the site was laid out using a common unit of measurement just over 30 centimeters in length - equal to about one modern-day foot. She also found evidence that some of the same geometrical constructs from Sun Temple were used in at least one other ancestral Puebloan ceremonial site, Pueblo Bonito, located in New Mexico's Chaco Culture National Historic Park.

"Further study is needed to see if that site also has the same common unit of measurement," she says. "It's a task that will keep us busy for some years to come."

The study "Advanced geometrical constructs in a Pueblo ceremonial site, c. 1200 CE" will appear in the Journal of Archaeological Science: Reports.

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Nicotine normalizes brain activity deficits that are key to schizophrenia

Work could lead to new non-addictive, nicotine-based treatments

A steady stream of nicotine normalizes genetically-induced impairments in brain activity associated with schizophrenia, according to new research involving the University of Colorado Boulder. The finding sheds light on what causes the disease and why those who have it tend to smoke heavily.

Ultimately the authors of the study, released online today in the journal Nature Medicine, envision their work could lead to new non-

addictive, nicotine-based treatments for some of the 51 million people worldwide who suffer from the disease.

"Our study provides compelling biological evidence that a specific genetic variant contributes to risk for schizophrenia, defines the mechanism responsible for the effect and validates that nicotine improves that deficit," said Jerry Stitzel, a researcher at the Institute for Behavioral Genetics (IBG) and one of four CU Boulder researchers on the study.

Led by Uwe Maskos -- a researcher at the Institut Pasteur in Paris, France -- the study found that when mice with schizophrenic characteristics were given nicotine daily, their sluggish brain activity increased within two days. Within one week it had normalized.

"Basically the nicotine is compensating for a genetically determined impairment," says Stitzel. "No one has ever shown that before."

The international team of scientists set out to explore the underlying causes of "hypofrontality" -- a reduction of neuronal firing in the prefrontal cortex of the brain. Hypofrontality is believed to be the root cause of many of the signature cognitive problems experienced by schizophrenics, including trouble paying attention, remembering things, making decisions and understanding verbal explanations.

Previous genome-wide association studies have suggested that people with a variation in a gene called CHRNA5 are more likely to have schizophrenia, but the mechanism for that association has remained unclear. People with that variant are also more likely to smoke.

Eighty to 90 percent of people with schizophrenia smoke and most are very heavy smokers, a fact that has long led researchers to suspect they are self-medicating.

For the study, the researchers set out to answer several questions: Does a variant in the CHRNA5 gene lead to hypofrontality. If so, how? And does nicotine somehow interrupt this effect?

To do so, the research team first took mice with the CHRNA5 gene variant and used state-of-the-art brain imaging technologies to see if they had hypofrontality. They did. Then Stitzel and co-author Charles Hoeffer, also of CU Boulder's IBG, conducted behavioral tests to see if the mice shared key characteristics of schizophrenics, like being unable to suppress a startle response and being averse to social interaction. They were. The results validated that the gene variant likely plays a role in schizophrenia by causing hypofrontality, says Stitzel.

Nicotine appeared to reverse this in the mice, normalizing brain activity by acting on nicotinic receptors in regions of the brain key to healthy cognitive function.

Because hypofrontality is also associated with addiction and other psychiatric conditions, such as attention deficit hyperactivity disorder and Bipolar disorder, the research could ultimately have broad applications for drug development in the mental health field, the authors say.

"This defines a completely novel strategy for medication development," says lead author Maskos. Early stage research is already under way to develop drugs that act on nicotinic receptors.

Another potential application of the research: "Identifying behavioral deficits associated with this mutation can be used for diagnostic or predictive work in schizophrenia," says Hoeffer.

Other co-authors included IBG research associates Josien Levenga and Heidi O'neill.

http://bit.ly/2jWBFqs

TSRI scientists create first stable semisynthetic organism

Scientists at have announced the development of the first stable semisynthetic organism

LA JOLLA, CA - Life's genetic code has only ever contained four natural bases. These bases pair up to form two "base pairs"--the rungs of the DNA ladder--and they have simply been rearranged to create bacteria and butterflies, penguins and people. Four bases make up all life as we know it.

Until now. Scientists at The Scripps Research Institute (TSRI) have announced the development of the first stable semisynthetic organism. Building on their 2014 study in which they synthesized a DNA base

pair, the researchers created a new bacterium that uses the four natural bases (called A, T, C and G), which every living organism possesses, but that also holds as a pair two synthetic bases called X and Y in its genetic code.

TSRI Professor Floyd Romesberg and his colleagues have now shown that their single-celled organism can hold on indefinitely to the synthetic base pair as it divides. Their research was published January 23, 2017, online ahead of print in the journal Proceedings of the National Academy of Sciences.

"We've made this semisynthetic organism more life-like," said Romesberg, senior author of the new study.

While applications for this kind of organism are still far in the future, the researchers say the work could be used to create new functions for single-celled organisms that play important roles in drug discovery and much more.

Building a Unique Organism

When Romesberg and his colleagues announced the development of X and Y in 2014, they also showed that modified E. coli bacteria could hold this synthetic base pair in their genetic code. What these E. coli couldn't do, however, was keep the base pair in their code indefinitely as they divided. The X and Y base pair was dropped over time, limiting the ways the organism could use the additional information possessed in their DNA.

"Your genome isn't just stable for a day," said Romesberg. "Your genome has to be stable for the scale of your lifetime. If the semisynthetic organism is going to really be an organism, it has to be able to stably maintain that information."

Romesberg compared this flawed organism to an infant. It had some learning to do before it was ready for real life.

In stepped TSRI Graduate Student Yorke Zhang and Brian Lamb, an American Cancer Society postdoctoral fellow in the Romesberg lab at the time of the study. Together, they helped develop the means for the single-celled organism to retain the artificial base pair.

First, Zhang and Lamb, co-first authors of the study, optimized a tool called a nucleotide transporter, which brings the materials necessary for the unnatural base pair to be copied across the cell membrane. "The transporter was used in the 2014 study, but it made the semisynthetic organism very sick," Zhang explained. The researchers discovered a modification to the transporter that alleviated this problem, making it much easier for the organism to grow and divide while holding on to X and Y.

Next, the researchers optimized their previous version of Y. The new Y was a chemically different molecule that could be better recognized by the enzymes that synthesize DNA molecules during DNA replication. This made it easier for cells to copy the synthetic base pair.

A New Use for CRISPR-Cas9

Finally, the researchers set up a "spell check" system for the organism using CRISPR-Cas9, an increasingly popular tool in human genome editing experiments. But instead of editing a genome, the researchers took advantage of CRISPR-Cas9's original role in bacteria.

The genetic tools in CRISPR-Cas9 (a DNA segment and an enzyme) originated in bacteria as a kind of immune response. When a bacterium encounters a threat, like a virus, it takes fragments of the invader genome and pastes them into its own genome--a bit like posting a "wanted" poster on the off chance it sees the invader again. Later, it can use those pasted genes to direct an enzyme to attack if the invader returns.

Knowing this, the researchers designed their organism to see a genetic sequence without X and Y as a foreign invader. A cell that dropped X and Y would be marked for destruction, leaving the scientists with an organism that could hold on to the new bases. It was like the organism was immune to unnatural base pair loss.

"We were able to address the problem at a fundamental level," said Lamb, who now serves as a research scientist at Vertex Pharmaceuticals.

Their semisynthetic organism was thus able to keep X and Y in its genome after dividing 60 times, leading the researchers to believe it can hold on to the base pair indefinitely.

"We can now get the light of life to stay on," said Romesberg. "That suggests that all of life's processes can be subject to manipulation."

A Foundation for Future Research

Romesberg emphasized that this work is only in single cells and is not meant to be used in more complex organisms. He added that the actual applications for this semisynthetic organism are "zero" at this point. So far, scientists can only get the organism to store genetic information.

Next, the researchers plan to study how their new genetic code can be transcribed into RNA, the molecule in cells needed to translate DNA into proteins. "This study lays the foundation for what we want to do going forward," said Zhang.

Additional authors of the study, "A semisynthetic organism engineered for the stable expansion of the genetic alphabet," were Aaron W. Feldman and Anne Xiaozhou Zhou of TSRI; Thomas Lavergne of the University of Grenoble; and Lingjun Li of Henan Normal University.

The study was supported by the National Institutes of Health (grant GM060005), a National Science Foundation Graduate Research Fellowship (grant DGE-1346837), the National Natural Science Foundation of China (grant 21472036), a Labex ARCANE grant (ANR-11-LABX-0003-01), NanoBio-ICMG platforms (FR 2607) and a postdoctoral fellowship from the American Cancer Society, Illinois Division.

http://bit.ly/2jTq01z

Hidden Heart Risks? Masked Hypertension May Affect 17 Million

Nearly one in eight Americans who think that they have normal blood pressure may have a type of high blood pressure that doesn't show up at the doctor's office, a new study finds.

By Sara G. Miller, Staff Writer | January 23, 2017 04:50pm ET

The phenomenon, called "masked hypertension," refers to a condition in which a person's blood pressure measurements are normal when taken in a doctor's office but elevated outside the office, during the individual's day-to-day activities, the study said.

People with masked hypertension may be at increased risk for heart disease, according to the study, published Jan. 18 in the American Journal of Epidemiology.

Masked hypertension is not the only condition in which people's blood pressure measurements appear to change between their doctor's office and the real world, the researchers wrote. Another phenomenon, known as "white-coat hypertension," also exists, but is the opposite of masked hypertension: People with this condition have high blood pressure when it is measured in the doctor's office, but normal blood pressure at other times.

Unlike masked hypertension, however, white-coat hypertension is not typically linked to an increased risk for heart disease, the study said.

To figure out how many Americans have masked hypertension, the researchers looked at two datasets. The first came from a study called the Masked Hypertension Study, and included more than 800 adults who did not have high blood pressure when it was measured in a doctor's office. At the beginning of the study, nurses or technicians manually measured each participant's blood pressure three times.

Then, to measure blood pressure outside of the doctors' offices, the participants wore 24-hour blood pressure monitors, the study said. These devices took blood pressure readings every 28 minutes over a 24-hour period.

Data from the 24-hour blood pressure monitors showed that about 14 percent of these participants had masked hypertension, the study said.

But the participants in the Masked Hypertension Study weren't representative of the entire U.S., so the researchers used data from that study along with data from large-scale survey: an ongoing national survey called the National Health and Nutrition Examination Study (NHANES). The data from NHANES includes information on demographics and blood pressure levels for people from across the U.S. From this, the researchers estimated that out of the nearly 140 million U.S. adults who are thought to have normal blood pressure,

about 12 percent, or 17 million people, actually have masked hypertension.

This means that doctors may fail to recognize that this large group of adults has an increased risk for heart disease, the researchers wrote in the study. The research was led by Dr. Claire Wang, an associate professor of health policy and management at the Mailman School of Public Health at Columbia University in New York City.

In addition, these individuals could benefit from treatments aimed at lowering blood pressure, the researchers said.

To properly diagnose masked hypertension, the researchers suggested using 24-hour blood pressure monitors. Similarly, the U.S. Preventive Services Task Force (USPSTF) recently recommended that doctors use these monitors to better identify patients who have white-coat hypertension, the researchers noted. The USPSTF organization makes recommendations regarding the effectiveness of preventive health services and also considers whether the benefits of treatments outweigh the risks.

The investigators in the new study noted that their research aims to provide only an "interim estimate" of the number of Americans who have masked hypertension. More research is needed to confirm the findings, the researchers wrote.

http://bit.ly/2km4803

Pre-eclampsia breakthrough 'to save lives'

Australian scientists have discovered a drug used to treat reflux has the potential to "wipe out" pre-eclampsia, a deadly pregnancy complication that affects thousands of women every year.

Sarah Wiedersehn, Australian Associated Press

Pre-eclampsia is a condition where the placenta releases toxins through the body that can damage blood vessels and lead to organ failure in pregnant women. There is no treatment other than to deliver the baby early, putting the child's life at risk.

But in a medical breakthrough, an effective and life-saving treatment could be available within the next five years, says Dr Natalie Hannan from the Translational Obstetrics Group (TOG) based at Melbourne's Mercy Hospital for Women.

Scientists at the hospital have shown that the drug esomeprazole - a proton pump inhibitor used to treat gastric reflux and indigestion - can switch off the production of toxins from the pre-eclamptic placenta.

The proton inhibitors were also successful in bringing down blood pressure - the main symptom of pre-eclampsia - in mice with the disease, according to the study published in medical journal Hypertension.

Dr Hannan, who led the international study, says she was "astonished" by the effectiveness of the drug. "We actually can't believe how great the drug is working to block these toxins."

It's estimated between 60,000 to 70,000 women are lost to pre-eclampsia each year globally and around half-a-million babies.

Already proven safe to use during pregnancy, a major clinical trial to test the drug is now underway in South Africa. A total of 120 pregnant women with the disease will be treated with esomeprazole at Tygerberg Hospital in Cape Town. Further trials in Australia are also planned. "If proton pump inhibitors can reduce the burden of pre-eclampsia, it could save the lives of thousands of mothers and babies globally," said Professor Stephen Tong, the head of TOG.

http://bit.ly/2jWN145

Sex toys 'safer' than children's toys, Swedish chemicals study finds

Of surveyed sex toys, 2% contained banned chemicals compared with 15% of children's toys tested a year before

Fewer sex toys than children's toys contain dangerous chemicals, according to a new report by a Swedish inspection authority.

In its study conducted in 2016, 2% of the 44 surveyed sex toys that had been imported to Sweden contained banned chemicals, the Swedish Chemicals Agency (SCA) said. In a separate study the year before, the agency tested 112 children's toys in Sweden and found 15% contained banned chemical substances, including lead.

"This was a bit surprising," Frida Ramstrom, an inspector for the agency, told AFP. "This was the first time we did such a study."

Of the 44 sex toys examined, only one plastic dildo was found to contain a banned substance: chlorinated paraffins, which is suspected of causing cancer, the SCA said. It said it was difficult to determine why more children's toys contained dangerous chemicals.

But one contributing factor was that sex toys were often imported by larger companies, which could exert more pressure on manufacturers to avoid harmful chemicals, whereas children's toys were more often imported by smaller companies which had less power to make such demands, according to Björn Malmström, a spokesman for the SCA.

Swedish law stipulates that chemicals in children's toys "must never pose a risk to human health".

Three of the 44 examined sex toys, made of artificial leather and bondage tape, contained a type of phthalates used as a plasticiser at levels above a 0.1% threshold, the agency said.

That specific type of phthalates is not banned in sex toys but is on the EU list of chemicals of "very high concern" as it can affect the body's hormonal balance and cause infertility. Companies are therefore required to inform consumers if a product contains more than 0.1%.

The global market for sex products is estimated at about $20bn (£16bn) a year, according to British market research group Technavio. It is expected to grow by nearly 7% a year between 2016 and 2020.

People in the US and China are among the biggest consumers of sex toys, according to Technavio.

http://bit.ly/2kANb22

Fighting Cancer's Crisis of Confidence, One Study at a Time

Every year the US government spends $5 billion on cancer research. That's not counting outgoing Vice President Joe Biden's $1 billion cancer moonshot, or private projects like Sean Parker's $250 million cancer immunotherapy institute. And yet more than 8 million people still die every year from the disease—despite the frequent refrain that a cure is just around the corner. Exhibit A: this 2003 WIRED article, "The End of Cancer."

It's a tempting prediction. Scientists today are exploring more promising new technologies than ever before: whole-genome sequencing, liquid biopsies, mRNA vaccines, AI-powered imaging analysis. But that doesn't necessarily mean they're more promising. No number of flashy new disruptors can fix cancer research's real problem: much of its data can't be trusted, because it was never validated. That's why a group called the Open Science Collaboration is reexamining the results from the 29 most important cancer papers of the last few years. Today, it published the first findings from its newest reproducibility project. Do they restore faith in the foundations of cancer research? Not exactly. But it is a start.

Revolutionizing Reproducibility

Scientists from two huge pharmaceutical companies, of all people, began signaling cancer's reproducibility crisis a little over five years ago. In 2011, a team from Bayer Healthcare reported when they tried to replicate the results of basic cancer studies—something drug companies do routinely to direct new drug development, they could only validate 25 percent of them. Shortly after, the former head of cancer research at Amgen published a paper in Nature saying that his scientists could only replicate six out of 53 "landmark" cancer studies. The reports shocked the cancer community, and kicked off a media storm questioning the legitimacy of cancer science—and science in general.

Brian Nosek saw this as a challenge. A University of Virginia psychologist, Nosek founded the Center for Open Science in 2011 to investigate the reproducibility of canonical psychology studies. But a few years later he turned his attention to cancer biology, and with $2 million from replication advocates John and Laura Arnold he assembled a team to reconstruct the individual experiments of the 50 most influential cancer biology papers published between 2010 and 2012.

It started out simply enough. Nosek enlisted microbiologist Tim Errington to manage the project and Elizabeth Iorns at the rent-a-researcher firm Science Exchange to farm out tasks to her network of 900 privately-contracted labs. They began emailing authors to collaborate on an approach to replicating the key findings of each paper, which ranged from investigations into prostate cancer-fighting microRNA to the impact of gut microbes in colon cancer. But that's where the project began to stumble. Cells and mice and peptides, it turned out, are difficult to share. You can't just go to the fridge and then pop down to your local Fed-Ex.

"The norm for these kinds of studies is to not include all the raw data or a detailed protocol," says Iorns. Instead, she and Errington had to track down that information from each of the original authors, a time-consuming process neither party much enjoyed. Not only did some of the researchers find the whole thing a nuisance, but sometimes the labs didn't even know who did what on the original paper, as graduate students or post-docs who did the bulk of the work had since moved on. "I was surprised how much institutions are not set up to support reproducibility," says Errington. "They're actually set up to protect materials against sharing." Losing more time and money to this process than expected, last summer the project downsized from 50 to 29 papers.

So far, the Center has completed seven of those studies, and eLife published the first five fully analyzed efforts today. In stark terms, one failed to replicate, two were inconclusive due to technical difficulties, and two generally supported the initial findings.

But Errington and Nosek caution that those surface judgments don't mean much. Mostly they just show how hard it is to interpret replication results. Studies can fail a reproducibility test for a number of reasons, none of which mean the original results are false. Teasing that out is where it gets interesting.

Controlling for Cancer

Take, for example Levi Garraway's 2012 paper about melanoma. Garraway and his team showed that a mutation called PREX2 sped up the growth of human tumor cells that were transplanted onto mice. The finding, originally published in Nature, has been cited 422 times, including in papers evaluating PREX2 as a potential target for small molecule therapies for skin cancer.

When Errington's replicating team tried the same experiment with the same strain of mice and melanoma cells from Garraway's lab at the Dana-Farber Cancer Institute, they found that the PREX2 mutations made no difference in tumor growth. But that's because all the mice started getting tumors within a week or two, regardless of whether they had the mutation. The team couldn't observe any differences, because their tumors were progressing at a turbo-charged speed.

Does that mean the study failed? Not really. Because the model didn't act the way they expected, Errington's team couldn't even test the hypothesis. The cells could have changed in the intervening years, or the mice could have been raised differently, or any number of other things could have happened. There's no way to know for sure.

Contrast that to a 2011 paper written by researchers at Stanford and the Lucille Packard Children's Hospital describing a computer model to predict if already-approved drugs would also be good at fighting cancers. They validated the approach by testing those drugs against controls on different kinds of tumors. When the replication team retested the same drugs on the same kinds of cancer cells, they found similar effects—but they weren't as significant as the original findings. Now the conversation researchers can have is about statistical cutoffs, not whether or not the model works. And that's a significant narrowing of scientific uncertainty.

Which is after all, what science is all about. "Humans love certainty, but the evidence rarely provides it," says Nosek. "Each study reduces the incompleteness of our understanding about the world. And we have to just do it over and over until we get to a place where we get consistent evidence."

So, five studies down, only a 4.5 million more to go. But who's counting?

http://bit.ly/2kaOK6e

Blinking Acts Like 'Steadicam of the Mind'

Blinking appears to serve a bigger function than just lubricating the eyeballs.

On average, people blink tens of thousands of times a day, and while it was thought the involuntary motion served mainly to lubricate the eyeballs, a new study suggests blinking has a more important role.

Writing in the journal Current Biology, an international team of researchers led by the University of California at Berkeley say blinking "repositions our eyeballs so we can stay focused on what we're viewing."

According to researchers, when we blink, our eyes roll back in their sockets, but they don't always return to the exact same position after the blink. This causes the brain to spur eye muscles to "realign our vision."

"Our eye muscles are quite sluggish and imprecise, so the brain needs to constantly adapt its motor signals to make sure our eyes are pointing where they're supposed to," said study lead author Gerrit Maus, an assistant professor of psychology at Nanyang Technological University in Singapore. "Our findings suggest that the brain gauges the difference in what we see before and after a blink, and commands the eye muscles to make the needed corrections."

Without these corrections, researchers say "our surroundings would appear shadowy, erratic and jittery," adding that the mechanism acts "like a steadicam of the mind."

To reach their conclusions, researchers say they conducted "the most boring experiment ever," in which participants sat in a dark room while staring at a dot on a screen. Using infrared cameras, the tracked blinks and eye movements.

After each blink, the dot was moved one centimeter to the right, something that was not noticed by the participants. However, the brain registered the dot's movement and triggered eye muscles to refocus on the dot.

After 30 times participants' eyes adjusted during each blink and shifted automatically to the spot where they predicted the dot to be."

"Even though participants did not consciously register that the dot had moved, their brains did, and adjusted with the corrective eye movement," Maus said. "These findings add to our understanding of how the brain constantly adapts to changes, commanding our muscles to correct for errors in our bodies' own hardware."

http://bbc.in/2jm7Mlk

Brazil sees sharp rise in yellow fever cases

Health officials in Brazil say there has been a sharp rise in the cases of yellow fever in the country.

They said there had been 63 confirmed cases of the mosquito-borne illness so far this year, up from seven in the whole of 2016.

Most of the cases have been in rural areas of Minas Gerais state, a Ministry of Health statement said. The government has sent two million doses of yellow fever vaccines to the state. The governor of Minas Gerais has declared a 180-day state of emergency.

What is yellow fever?

Caused by a virus that is transmitted to humans by mosquitoes

Difficult to diagnose and often confused with other diseases or fevers

Most people recover after the first phase of infection that usually involves fever, muscle and back pain, headache, shivers, loss of appetite, and nausea or vomiting

About 15% of people face a second, more serious phase involving high fever, jaundice, bleeding and deteriorating kidney function

Half of those who enter the "toxic" phase usually die within 10 to 14 days

Source: WHO

Of the 63 confirmed cases in Brazil, 35 have proved fatal, Brazilian Health Ministry figures show. That is the highest number of deaths since at least 2008, the year to which Ministry of Health records date back.

There have also been three confirmed cases in Sao Paulo, Brazil's most populous state, and one each in Espiritu Santo and Bahia, which both neighbour Minas. It is not clear what has caused the rise in cases.

http://bit.ly/2k9QMmr

Can mushrooms help delay or prevent dementia and Alzheimer's disease?

Presenting evidence supporting a potential role of mushrooms to reduce or delay development of age-related neurodegeneration

New Rochelle, NY - Certain edible and medicinal mushrooms contain bioactive compounds that may enhance nerve growth in the brain and protect against neurotoxic stimuli such as inflammation that contribute to neurodegenerative diseases like dementia and Alzheimer's disease. The evidence supporting a potential role of mushrooms as functional foods to reduce or delay development of age-related neurodegeneration is presented in an article published in Journal of Medicinal Food, a peer-reviewed journal from Mary Ann Liebert, Inc., publishers. The article is available free on the Journal of Medicinal Food website until February 24, 2017.

In "Edible and Medicinal Mushrooms: Emerging Brain Food for the Mitigation of Neurodegenerative Diseases," Chia Wei Phan, Pamela David, and Vikineswary Sabaratnam, University of Malaya, Kuala Lumpur, Malaysia, discuss the scientific findings related to the health benefits of edible and culinary mushrooms. The authors focus on the activity of bioactive components of mushrooms that may offer neuroprotective and cognitive benefits.

"In contrast to the body of literature on food ingredients that may benefit cardiometabolic diseases and cancer, very few studies have focused on food that may benefit neurodegenerative diseases," says Journal of Medicinal Food Editor-in-Chief Sampath Parthasarathy, MBA, PhD, Florida Hospital Chair in Cardiovascular Sciences and Interim Associate Dean, College of Medicine, University of Central Florida. "The current study might stimulate the identification of more food materials that are neuroprotective."

http://bit.ly/2jESvx8

Antibiotics, not dirty hospitals, the main cause of C. difficile epidemic

Overuse of antibiotics like ciprofloxacin led to the outbreak of severe diarrhoea caused by C. difficile

The study concluded that overuse of antibiotics like ciprofloxacin led to the outbreak of severe diarrhoea caused by C. difficile that hit headlines from 2006 onwards. The outbreak was stopped by substantially reducing use of ciprofloxacin and related antibiotics.

Inappropriate use and widespread over prescribing of fluoroquinolone antibiotics such as ciprofloxacin in fact allowed C. difficile bugs that were resistant to the drug to thrive, because non-resistant bugs in the gut were killed off by the antibiotic, leaving the way clear for rapid growth of resistant C. difficile. Concerns about hospital "superbugs" which had become resistant to common antibiotics resulted in the announcement of a programme of "deep cleaning" and other infection control measures in the NHS in 2007.

The study, by the University of Oxford, University of Leeds and Public Health England and published today in The Lancet Infectious Diseases, found that cases of C. difficile fell only when fluoroquinolone use was restricted and used in a more targeted way as one part of many efforts to control the outbreak.

The restriction of fluoroquinolones resulted in the disappearance in the vast majority of cases of the infections caused by the antibiotic-resistant C. difficile, leading to around an 80% fall in the number of these infections in the UK (in Oxfordshire approximately 67% of C. difficile bugs were antibiotic-resistant in September 2006, compared to only approximately 3% in February 2013).

In contrast, the smaller number of cases caused by C. difficile bugs that were not resistant to fluoroquinolone antibiotics stayed the same. Incidence of these non-resistant bugs did not increase due to patients being given the antibiotic, and so were not affected when it was restricted.

At the same time, the number of bugs that were transmitted between people in hospitals did not change. This was despite the implementation of comprehensive infection prevention and control measures, like better handwashing and hospital cleaning in this case.

The study's authors therefore conclude that ensuring antibiotics are used appropriately is the most important way to control the C. difficile superbug. The authors note that it is important that good hand hygiene and infection control continues to be practiced to control the spread of other infections.

The study, analysed data on the numbers of C. diff infections and amounts of antibiotics used in hospitals and by GPs in the UK.

More than 4,000 C. diff bugs also underwent genetic analysis using a technique called whole genome sequencing, to work out which antibiotics each bug was resistant to.

Co-author Derrick Crook, Professor of Microbiology, University of Oxford said: "Alarming increases in UK hospital infections and fatalities caused by C. difficile made headline news during the mid-2000s and led to accusations of serious failings in infection control.

"Emergency measures such as 'deep cleaning' and careful antibiotic prescribing were introduced and numbers of C. difficile infections gradually fell by 80% but no-one was sure precisely why.

"Our study shows that the C. difficile epidemic was an unintended consequence of intensive use of an antibiotic class, fluoroquinolones, and control was achieved by specifically reducing use of this antibiotic class, because only the C. difficile bugs that were resistant to fluoroquinolones went away.

"Reducing the type of antibiotics like ciprofloxacin was, therefore, the best way of stopping this national epidemic of

C. difficile and routine, expensive deep cleaning was unnecessary. However it is important that good hand hygiene continues to be practiced to control the spread of other infections.

"These findings are of international importance because other regions such as North America, where fluoroquinolone prescribing remains unrestricted, still suffer from epidemic numbers of C. difficile infections."

Co-author Prof Mark Wilcox, Professor of Microbiology, University of Leeds, said: "Our results mean that we now understand much more about what really drove the UK epidemic of C. diff infection in the mid-2000s. "Crucially, part of the reason why some C. diff strains cause so many infections is because they find a way to exploit modern medical practice.

Similar C. diff bugs that affected the UK have spread around the world, and so it is plausible that targeted antibiotic control could help achieve large reductions in C. diff infections in other countries."

The funding for the study came from the UK Clinical Research Collaboration, (Medical Research Council, Wellcome Trust, National Institute for Health Research); NIHR Oxford Biomedical Research Centre; NIHR Health Protection Research Unit in Healthcare Associated Infections and Antibiotic Resistance, University of Oxford in partnership with Leeds University and Public Health England; NIHR Health Protection Research Unit in Modelling Methodology, Imperial College London in partnership with Public Health England; and the Health Innovation Challenge Fund.

The paper will be available from 2330 GMT on 24 January 2017 at: http://www.thelancet.com/journals/laninf/article/PIIS1473-3099(16)30514-X/fulltext

http://bit.ly/2kDSZYF

Researchers discover BRCA1 gene is key for blood forming stem cells

BRCA1 gene is required for the survival of blood forming stem cells

DALLAS - Researchers at from the Harold C. Simmons Comprehensive Cancer Center have found that the BRCA1 gene is required for the survival of blood forming stem cells, which could explain why patients with BRCA1 mutations do not have an elevated risk for leukemia. The stem cells die before they have an opportunity to transform into a blood cancer.

"One of the great mysteries in cancer research is why inherited mutations, such as those in BRCA1, cause cancer only in specific tissues such as the breast and ovaries, rather than in all tissues. Our data suggest a 'die or transform' hypothesis, which could explain this tissue specificity," said Dr. Theodora Ross, Professor of Internal Medicine and Director of the Cancer Genetics Program at UT Southwestern. Additional data from this study suggest these patients may have a tougher time with the side effects of chemotherapy.

"Patients with certain BRCA1 mutations may be at a higher than expected risk for serious complications during chemotherapy treatment," said Dr. Ross, who holds the Jeanne Ann Plitt Professorship in Breast Cancer Research and the H. Ben and Isabelle T. Decherd Chair in Internal Medicine in Honor of Henry M. Winans, Sr., M.D. "If we confirm these clinical findings in upcoming studies, giving patients preventative antibiotics or growth factors may be necessary to lower this increased risk of treatment side effects."

The study is published in Cell Reports.

According to the National Cancer Institute, more than 246,660 women will be diagnosed with breast cancer and 22,280 women will be diagnosed with ovarian cancer this year. Of these, about 10-15 percent are estimated to be affected by BRCA1 and BRCA2 genetic mutations, Dr. Ross said.

"Our data also illustrate why rare variations in the BRCA1 gene are not always mutations that put women and men at high risk for specific cancers. We and others have learned that most rare 'spellings' of the BRCA1 gene, spellings we call variations of undetermined significance, are not harmful," said Dr. Ross, a member of the Simmons Cancer Center.

Dr. Ross' laboratory investigates how cells transform from normal cells to cancer cells, and how some cancer cells are able to withstand specifically targeted cancer drugs. Among her main avenues of research is the BRCA1 gene, which - when abnormal - predisposes women to breast and ovarian cancer.

This study was supported by a Burroughs Wellcome Fund Clinical Scientist Award in Translational Research to Dr. Ross, the National Institutes of Health, the National Cancer Institute, and the Cancer Prevention and Research Institute of Texas (CPRIT).

http://bit.ly/2jzj3SG

The folds in your brain may be linked to how neurotic you are

Those folds might be revealing

By New Scientist staff and Press Association

What are you like? A look at your brain may tell you. A study has found a link between some elements of brain structure and certain personality traits.

The study involved scanning the brains of 500 volunteers, and assessing their personalities in terms of five traits – neuroticism, openness, extraversion, agreeableness, and conscientiousness.

The researchers focused on the structure of the cortex, the outer layer of the brain.

They found that in people who are more neurotic and prone to mood changes, the cortex tends to be thicker and less wrinkly. People who appear more open – for example, curious and creative – show the opposite pattern.

More mature

The link between structure and personality may help explain how we mature as we get older.

Folds and wrinkles are thought to increase the surface area of the brain, but make the cortex thinner.

The cortex continues to stretch and fold throughout childhood and adolescence, and into adulthood.

As we grow up, people generally become less neurotic, and more conscientious and agreeable.

“Our work supports the notion that personality is, to some degree, associated with brain maturation,” says Roberta Riccelli, at Magna Graecia University in Catanzaro, Italy.

http://bit.ly/2jfGP7S

Researcher proposes novel mechanism to stop tsunamis in their tracks

Cardiff University mathematician believes tsunami energy can be dissipated using deep-ocean sound waves

Devastating tsunamis could be halted before hitting the Earth's shoreline by firing deep-ocean sound waves at the oncoming mass of water, new research has proposed.

Dr Usama Kadri, from Cardiff University's School of Mathematics, believes that lives could ultimately be saved by using acoustic-gravity waves (AGWs) against tsunamis that are triggered by earthquakes, landslides and other violent geological events. AGWs are naturally occurring sounds waves that move through the deep ocean at the speed of sound and can travel thousands of metres below the surface.

AGWs can measure tens or even hundreds of kilometres in length and it is thought that certain lifeforms such as plankton, that are unable to swim against a current, rely on the waves to aid their movement, enhancing their ability to find food.

In a paper published in the journal Heliyon, Dr Kadri proposes that if we can find a way to engineer these waves, they can be fired at an incoming tsunami and will react with the wave in such a way that reduces its amplitude, or height, and causes its energy to be dissipated over a large area. By the time the tsunami reaches the shoreline, Dr Kadri writes, the reduced height of the tsunami would minimise the damage caused to both civilians and the environment.

Dr Kadri also believes that this process of firing AGWs at a tsunami could be repeated continuously until the tsunami is completely dispersed. "Within the last two decades, tsunamis have been responsible for the loss of almost half a million lives, widespread long-lasting destruction, profound environmental effects and global financial crisis," Dr Kadri said. "Up until now, little attention has been paid to trying to mitigate tsunamis and the potential of acoustic-gravity waves remains largely unexplored."

The devastating tsunami that was generated in the Indian Ocean in 2004 after a magnitude 9 earthquake has been recorded as one of the deadliest natural disasters in recent history after it caused over 230,000 deaths in 14 countries. The energy released on the Earth's surface by the earthquake and subsequent tsunami was estimated to be the equivalent of over 1,500 times that of the Hiroshima atomic bomb.

In order to use AGWs in tsunami mitigation, engineers will firstly need to devise highly accurate AGW frequency transmitters or modulators, which Dr Kadri concedes would be challenging.

It may also be possible to utilise the AGWs that are naturally generated in the ocean when a violent geological event, such as an earthquake, occurs - essentially using nature's natural processes against itself. Indeed, Dr Kadri has already shown that naturally occurring AGWs could be utilised in an early tsunami detection system by placing detection systems in the deep ocean.

Dr Kadri continued: "In practice, generating the appropriate acoustic-gravity waves introduces serious challenges due to the high energy required for an effective interaction with a tsunami. However, this study has provided proof-of-concept that devastating tsunamis could be mitigated by using acoustic-gravity waves to redistribute the huge amounts of energy stored within the wave, potentially saving lives and billions of pounds worth of damage."

http://bit.ly/2jznP2x

Engineers develop 'smart glasses' that automatically focus on what wearer sees

The days of wearing bifocals or constantly swapping out reading glasses might soon come to an end.

A team led by University of Utah electrical and computer engineering professor Carlos Mastrangelo and doctoral student Nazmul Hasan has created "smart glasses" with liquid-based lenses that can automatically adjust the focus on what a person is seeing, whether it is far away or close up.

Research on the adaptive lenses was published this week in a special edition of the journal, Optics Express.

The paper was co-authored by U electrical and computer engineering associate professor Hanseup Kim and graduate researcher Aishwaryadev Banerjee.

"Most people who get reading glasses have to put them on and take them off all the time," says Mastrangelo, who also is a professor for USTAR, the Utah Science Technology and Research economic development initiative. "You don't have to do that anymore. You put these on, and it's always clear."

Early prototype of 'smart glasses' with liquid-based lenses that can automatically adjust the focus on what a person is seeing, whether it is far away or close up. The lenses are placed in battery-powered frames that can automatically adjust the focal length of the lenses based on what the wearer is looking at. Researchers expect to have smaller, lighter frames with the technology in as early as three years. Credit: **Dan Hixson/University of Utah College of Engineering**

The human eye has a lens inside that adjusts the focal depth depending on what you look at.

But as people age, the lens loses its ability to change focus, which is why many people ultimately require reading glasses or bifocals to see objects up close and regular eyeglasses to see far away, also known as farsightedness and nearsightedness, respectively.

So Mastrangelo and Hasan have created eyeglass lenses made of glycerin, a thick colorless liquid enclosed by flexible rubber-like membranes in the front and back.

The rear membrane in each lens is connected to a series of three mechanical actuators that push the membrane back and forth like a transparent piston, changing the curvature of the liquid lens and therefore the focal length between the lens and the eye.

"The focal length of the glasses depends on the shape of the lens, so to change the optical power we actually have to change the membrane shape," Mastrangelo says.

The lenses are placed in special eyeglass frames also invented by Mastrangelo, Hasan and other members of the research group with electronics and a battery to control and power the actuators.

In the bridge of the glasses is a distance meter that measures the distance from the glasses to an object via pulses of infrared light. When the wearer looks at an object, the meter instantly measures the distance and tells the actuators how to curve the lenses.

If the user then sees another object that's closer, the distance meter readjusts and tells the actuators to reshape the lens for farsightedness.

Hasan says the lenses can change focus from one object to another in 14 milliseconds. A rechargeable battery in the frames could last more than 24 hours per charge, Mastrangelo says.

Before putting them on for the first time, all users have to do is input their eyeglasses prescription into an accompanying smartphone app, which then calibrates the lenses automatically via a Bluetooth connection.

Users only needs to do that once except for when their prescription changes over time, and theoretically, eyeglass wearers will never have to buy another pair again since these glasses would constantly adjust to their eyesight.

Currently, the team has constructed a bulky working prototype that they put on display at last month's Consumer Electronics Show in Las Vegas, but expect to constantly improve the design to make them smaller and lighter.

Mastrangelo said a lighter, more attractive pair could hit the marketplace in as early as three years and that a startup company, Sharpeyes LLC, has been created to commercialize the glasses.

http://bit.ly/2jHhv6T

Triggering the brain's auto-focus

Researchers pinpoint brain structure that links environmental cues to enhanced focus

DURHAM, N.C. - We are constantly being bombarded with attention-grabbing distractions, from the flashy shopfronts and advertisements that flank the side of the road to the tempting buzz of the phone during a meeting with the boss.

For a long time, brain scientists believed that maintaining focus in these situations required a cascade of mental events: momentary distraction, followed by the realization that our attention has been diverted and a conscious effort to bring it back on the task at hand.

But recent research shows that our brains actually have a clever mechanism for outwitting these distractions. With repeated practice, environmental cues -- such as a particularly busy intersection, or your boss's office -- can trigger the brain to jump directly into a more focused state, bypassing distraction and saving precious time.

In a new study, Duke University researchers combined brain imaging with a celebrity naming game to pinpoint the structure in the brain responsible for forming direct links between environmental stimuli and enhanced focus. This structure, called the caudate nucleus, is also known to play an important role in linking motor actions to their consequences.

The brain's ability to automatically trigger greater focus was first demonstrated in 2011, and little is understood about how it handles this process, said senior author Tobias Egner, associate professor of psychology and neuroscience at Duke.

To unravel the neural mechanisms involved, Egner and postdoctoral researcher Yu-Chin Chiu asked research subjects to carry out a variation on a classic test of mental control, called the Stroop test, while their brain activity was monitored with functional magnetic resonance imaging (fMRI).

In a twist on the test, the 28 participants were shown a series of images of famous Hollywood men, including Brad Pitt, Tom Cruise, Matt Damon and Leonardo DiCaprio, and asked to identify each actor as quickly as they could. To make the task more challenging, written names appeared across each actors' faces in bold red ink, and the name and photo correctly matched only half of the time.

People usually take longer to respond when the names and the photos don't match, Egner said, because they have to overcome their initial impulse to read the name and instead direct their full focus on the face.

But the researchers found that if they frequently paired a specific face with incorrect names -- for example, Leonardo DiCaprio's face with the names Pitt, Cruise, or Damon -- participants became better at identifying the face and ignoring the names.

Since there is no way of knowing in advance which face will appear, or if the name will match, the only way to get faster is if the face itself directly triggers the brain to focus on the face and ignore the name, said Egner, who is also a member of Duke's Institute for Brain Sciences.

"If 75% of the time Leonardo DiCaprio appears with other people's names, you learn that when you see DiCaprio's face, you should ignore the distracting word," Egner said.

As the research subjects learned to associate specific faces with a greater focus, fMRI scans showed a structure called the caudate nucleus lighting up. "It seems that the caudate nucleus doesn't just link motor actions to outcomes but also perhaps cognitive actions to outcomes," Egner said. "In this case, what is the correct attentional focus to make me perform well on this task?"

The results highlight how the brain's ability to form well-learned, reflexive reactions -- like reaching for the phone when it buzzes -- is not always at odds with controlled, or intentional behavior, and in fact the two processes can actually work together.

"There is a very longstanding tradition in psychology of distinguishing between automatic or really well-learned, well-practiced behavior and

intentional, controlled behavior," Egner said. "Here they really become partners in helping the performance."

"This work is exciting in that it highlights a brain-based mechanism that supports a human's use of reactive control -- the rapid and flexible deployment of attention to reduce susceptibility to distraction," said Julie Bugg, assistant professor of psychological and brain sciences at Washington University who was not an author on the study. "It captures the intimate relationship between learning and cognitive control." This work appears Jan. 25 in The Journal of Neuroscience.

CITATION: "The Caudate Nucleus Mediates Learning of Stimulus-Control State Associations," Yu-Chin Chiu, Jiefeng Jiang and Tobias Egner. The Journal of Neuroscience, Jan 25, 2017. DOI: # 10.1523/JNEUROSCI.0778-16.2017

http://bit.ly/2kcR2kK

Antibody combination puts HIV on the ropes

Combination of three antibodies from an elite controller suppresses HIV in mice

Without antiretroviral drug treatment, the majority of people infected with HIV ultimately develop AIDS, as the virus changes and evolves beyond the body's ability to control it. But a small group of infected individuals--called elite controllers--possess immune systems capable of defeating the virus. They accomplish this by manufacturing broadly neutralizing antibodies, which can take down multiple forms of HIV.

Now a study using antibodies from one of these elite controllers has shown that a combination of three such antibodies can completely suppress the virus in HIV-infected mice. The findings, from the laboratory of Michel Nussenzweig, who is Zanvil A. Cohn and Ralph M. Steinman Professor at Rockefeller University and head of the Laboratory of Molecular Immunology, are being reported in Science Translational Medicine.

"Some people with HIV produce these antibodies, but most of the time the virus eventually escapes them through mutations in the antibody's corresponding epitope," says postdoctoral fellow Natalia Freund, the study's first author. The epitope is the part of the virus that antibodies recognize and attach themselves to, and this ability to mutate makes HIV particularly tricky to tame. It ensures that once the virus is in their bodies, people remain infected forever, and this may be the biggest roadblock in developing immune therapies to overcome the virus.

Tug of war

"Think of the relationship between the antibodies and the virus as an arms race that goes on and on," Freund says. "By mutating, some of the virus may escape the antibodies and continue growing. Years later, the body may produce new broadly neutralizing antibodies against the escaped virus, which in turn may mutate and escape yet again."

"What we've shown in this study is that after several rounds of escape from these particular antibodies, the virus seems to run out of options," she adds. "In this particular case, HIV eventually loses this arms race."

An elite controller's immune system can defeat the virus by coming up with new broadly neutralizing antibodies, and also by producing cytotoxic T cells--immune cells that can recognize and destroy infected cells to immobilize the virus. The patient whose HIV response created antibodies for the study has been working with the Rockefeller team for ten years, contributing his blood serum for their research. He was infected at least three decades ago, and has developed three different types of broadly neutralizing antibodies that bind to three different sites on the virus.

The remarkable thing about his antibodies is that they seem to complement each other's activity, completely shutting down HIV.

The investigators gave the three antibodies, called BG18, NC37, and BG1, to HIV-infected mice whose immune systems had been modified to more closely resemble those of humans. They found that the trio rendered the virus undetectable in two-thirds of the mice three weeks after it was administered.

"This study validates the approach of using three different antibodies to control HIV infection," Freund concludes, "pointing the way toward a potential new treatment for people infected with HIV."

http://bit.ly/2kDNhSa

Therapeutic hypothermia offers no significant benefits for infants or children after in-hospital car

Body cooling, or therapeutic hypothermia, is no more effective than actively keeping the body at a normal temperature

WHAT: In a multicenter, international study of infants and children who suffered cardiac arrest while in the hospital, NIH-funded researchers have found that body cooling, or therapeutic hypothermia, is no more effective than actively keeping the body at a normal temperature, or therapeutic normothermia.

The study is the first to look exclusively at in-hospital cardiac arrests in infants and children in order to compare the two temperature treatments. Earlier trials involving adults who went into cardiac arrest outside of a hospital had found that therapeutic hypothermia improved survival and brain function. However, recent trials in adults and children did not find such improvements when compared with patients whose temperature was actively maintained in a normal temperature range to prevent fever.

Current guidelines recommend either treatment for out-of-hospital cardiac arrests in children, as both methods have resulted in equal rates of survival and prevention of brain injury. But out-of-hospital cardiac arrests have different causes and outcomes than in-hospital ones, and the findings of the new study could inform new guidelines for treatment of the latter.

The study included 329 patients between the ages of 2 days and 18 years old who had sustained cardiac arrest while in a hospital. The researchers randomly divided them into two groups and found that children in the group treated with therapeutic hypothermia had the same survival rates and neurobehavioral functioning a year later as those treated by keeping the body at normal temperature.

The research--funded by the National Heart, Lung, and Blood Institute (NHLBI), part of the National Institutes of Health--was presented at the annual meeting of the Society for Critical Care Medicine and published simultaneously in the January 24 issue of the New England Journal of Medicine.

The study is part of the Therapeutic Hypothermia after Pediatric Cardiac Arrest (THAPCA) trials, the largest examination to date of therapeutic hypothermia in children other than newborns for any health condition.

WHO: Jonathan R. Kaltman, M.D., medical officer in the Heart Development and Structural Diseases Branch in the Division of Cardiovascular Sciences at the National Heart, Lung, and Blood Institute (NHLBI), is available to comment.

ARTICLE: F Moler et al. Therapeutic Hypothermia After In-Hospital Pediatric Cardiac Arrest in Children. New England Journal of Medicine. DOI: 10.1056/NEJMoa1610493

http://bit.ly/2kcMBX9

Young girls less likely to attribute brilliance to their own gender

Six-year-old girls are less likely than boys to believe that members of their gender are brilliant

Six-year-old girls are less likely than boys to believe that members of their gender are brilliant, reports a new study, which also found that girls at this age are more likely to shy away from activities said to be for children who are "really, really smart."

The results demonstrate a worrisome trend, given that career aspirations of young men and women are shaped by societal stereotypes about gender. To gain more insights into gender stereotypes, Lin Bian and colleagues set up a series of experiments with children age 5, 6 and 7 - stages when stereotypes are known to begin.

In one task, the children were told a brief story about a person who was "really, really smart," but no hints as to the protagonist's gender were provided. At age 5, both boys and girls were equally likely to choose their own gender as "really, really smart," yet by age 6 and 7, girls were girls were significantly less likely than boys to associate brilliance with their own gender.

In another set of questionnaires, children had to guess which of four children, two boys and two girls, "gets the best grades in school." In

contrast with the drop in brilliance scores, there was no significant difference between younger and older girls in the likelihood of selecting other girls as having top grades. Thus girls' perceptions of school achievement were separate from their perceptions of brilliance. Lastly, children were introduced to two novel games, one said to be for "children who are really, really smart" and the other for "children who try really, really hard." The researchers found no difference between game choice of boys and girls at age 5, but by age 6 and 7 girls were less interested than boys in the game for smart children, but not in the game for hard-working children.

http://bit.ly/2joZ8CD

Quick-and-dirty DNA repair sets the stage for smoking-related lung cancer

Stem cells proliferating most in response to damage caused by cigarette smoke repair their DNA using an error- prone process

The stem cells that proliferate the most in response to damage caused by cigarette smoke repair their DNA using a process prone to errors, setting the stage for lung cancer, according to a study publishing January 26, 2017 in the open-access journal PLOS Biology by Marie-Liesse Asselin-Labat and her team of the Walter and Eliza hall Institute of Medical Research, Australia.

Smoking is a strong risk factor for squamous cell carcinoma, the second most common form of lung cancer, but the relative contributions to carcinogenesis of two types of lung stem cells (basal cells and alveolar progenitor cells) --and the molecular reasons for accumulation of the DNA damage that leads to cancer -- have not been clear. To explore these issues, the authors isolated both types of cells from the lungs of heavy smokers and compared the activity of their genes, their rates of cell division, and their ability to repair their DNA in response to damage.

They found that carcinoma cells from smokers carried the "transcriptional fingerprint" (i.e. the pattern of genes that were switched on or off) of basal cells, suggesting that the tumors may originate from this type of cell. They showed that basal cells were much more efficient at repairing DNA damage, allowing them to survive and reproduce following exposure to chemicals or radiation that damaged the DNA. However, the principal repair pathway used by basal cells, called "non-homologous end joining," introduced many errors into the DNA. The combination of rapid but error-prone repair, the authors suggest, leads basal cells to accumulate a high burden of mutations that ultimately leads to carcinoma.

"Our results indicate that targeting DNA repair processes may be a promising approach to preventing and treating this form of lung cancer," said Asselin-Labat.

In your coverage please use this URL to provide access to the freely available article in PLOS Biology: http://dx.plos.org/10.1371/journal.pbio.2000731

Citation: Weeden CE, Chen Y, Ma SB, Hu Y, Ramm G, Sutherland KD, et al. (2017) Lung Basal Stem Cells Rapidly Repair DNA Damage Using the Error-Prone Nonhomologous End-Joining Pathway. PLoS Biol 15(1): e2000731. doi:10.1371/journal.pbio.2000731

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http://bit.ly/2jHvdXx

Cluster of Mysterious Amnesia Cases Puzzles Researchers

No clear cause of their memory loss, leaving researchers puzzled about what exactly could have been behind these cases

By Rachael Rettner, Senior Writer | January 26, 2017 06:50pm ET

More than a dozen people in Massachusetts suddenly developed severe amnesia, but there was no clear cause of their memory loss, leaving researchers puzzled about what exactly could have been behind these cases, according to a new report.

In the report, researchers describe a cluster of 14 cases involving people who experienced sudden amnesia, and who were treated in Massachusetts from 2012 to 2016. All of the patients were relatively young (19 to 52 years old), and all had either tested positive for drugs or had a history of substance abuse, according to the report.

The patients showed "striking anterograde amnesia," according to the report, from researchers at the Lahey Hospital & Medical Center in

Burlington, Massachusetts, and the Massachusetts Department of Public Health (MDPH). People with this type of amnesia have trouble forming new memories, and they often cannot recall events from the immediate past, such as something that has just happened to them.

In nine of the cases, the patients were unconscious at the time they were brought to the hospital, and they experienced amnesia when they regained consciousness. In the five other cases, family members or friends noticed that the individuals were experiencing severe memory loss, and they brought them to the emergency room.

Twelve of the patients had a history of using opioid drugs, including prescription painkillers or heroin. Many of the patients had also used other drugs, including marijuana, cocaine and amphetamines. These drugs have many negative effects on people's health, but they typically have not been linked with the development of anterograde amnesia.

Notably, the brain scans of all of the patients revealed an unusual finding: MRI tests showed significantly reduced blood flow to a part of the brain called the hippocampus, which is important for memory formation. Humans have two hippocampi, one on either side of their brain, and the patients in the report had reduced blood flow to both hippocampi. But the researchers could not find a clear cause of this problem.

Sudden amnesia that's tied to reduced blood flow to both hippocampi is rare, the researchers said. A few similar cases have been reported in the past, but these were stand-alone cases rather than clusters of cases, the researchers noted. In some of those earlier cases, the sudden amnesia was tied to exposure to a toxic substance, such as carbon monoxide, the report said.

Investigation of the current cases is ongoing, and health authorities need to continue to watch for more cases, to determine whether this new cluster "represents an emerging syndrome related to substance use or other causes," such as exposure to a toxic substance, the researchers wrote in the Jan. 27 issue of the journal Morbidity and Mortality Weekly Report, which is published by the Centers for Disease Control and Prevention.

The cluster was first discovered in November 2015, when a neurologist in the Boston area reported four cases of unusual amnesia he had seen during the last three years. The MDPH conducted a subsequent search for similar cases and discovered the 10 additional ones.

Information on the long-term outcome of the cases was available for just four patients. Of these, one person's memory problems were resolved after five months, but two others continued to experience cognitive problems more than a year later. One of the patients still had severe short-term memory problems after eight weeks, and later died from cardiac arrest, the report said.

Doctors should consider performing an MRI and screening for drugs in all adult patients who have sudden-onset amnesia, the researchers said. Advanced laboratory testing, including testing for substances that were not assessed in the current report, might clarify why these cases were linked with drug use, they said.

http://bit.ly/2joLKic

Scientists develop new flu vaccines for man's best friend

It's that dreaded time of year - flu season. And we humans aren't the only ones feeling the pain. Dogs can get the flu, too.

Scientists at the University of Rochester School of Medicine and Dentistry have developed, for the first time, two new vaccines for canine influenza. This research is not only important for improving the health of our furry friends, but for keeping us safe, too. Dogs that have been infected with multiple influenza viruses have the potential to act as "mixing vessels" and generate new flu strains that could infect people. This hasn't happened yet, but experts say it's possible.

Today, veterinarians use vaccines that include inactivated or killed flu virus, but experts say they provide short-term, limited protection.

Scientists led by Luis Martinez-Sobrido, Ph.D., associate professor in the department of Microbiology and Immunology created two "live-

attenuated" vaccines against H3N8 canine influenza virus, which is currently circulating in dogs in the U.S. Past research shows that live-attenuated vaccines, made from live flu virus that is dampened down so that it doesn't cause the flu, provide better immune responses and longer periods of protection.

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University of Rochester School of Medicine and Dentistry

Martinez-Sobrido's team, including postdoctoral fellows Aitor Nogalez-Gonzalez, Ph.D. and Laura Rodriguez, Ph.D. used a genetic engineering technique called reserve genetics to create a live vaccine that replicates in the nose, but not in the lungs. The nose is where the virus first enters a dog's body, so generating an immune response there could stop the virus in its tracks. If the vaccine were to get into the lungs it could create unwanted inflammation in response to the live virus. The study, published in the Journal of Virology, found the live vaccine was safe and able to induce better immune protection against H3N8 canine influenza virus in mice and dog tracheal cells than a commercially available inactivated vaccine.

In a second study highlighted in the journal Virology the team used reserve genetics to remove a protein called NS1 from H3N8 canine influenza virus. Previous studies have shown that deleting the NS1 viral protein significantly weakens flu viruses so that they elicit an immune response but don't cause illness. This approach has been used with human, swine and equine flu viruses to generate potential vaccines and was also safe and more effective than a traditional inactivated H3N8 influenza vaccine in mice and dog tracheal cells.

Both studies were performed in collaboration with Collin Parrish, professor of Virology at the College of Veterinary Medicine at Cornell University and Pablo Murcia, professor at the University of Glasgow Centre for Virus Research.

The team is planning to test both live-attenuated vaccine approaches in clinical trials with dogs. The hope is to come up with new options to stem the spread of flu in shelters and kennels, and to avoid the transmission of a dog flu virus to people. As many dog owners and animal lovers are in close contact with dogs on a regular basis, Martinez-Sobrido believes its best to prevent dogs from getting the flu in the first place.

The team is using this research to tackle other dog flu viruses, too. They've used the safety of these approaches to engineer a live-attenuated vaccine for the H3N2 canine influenza virus, which was introduced in the United States in 2015. Early results show that similar to the H3N8 vaccine, the H3N2 live-attenuated vaccine is able to protect against the H3N2 canine influenza virus and is more effective than the only currently available inactivated vaccine.

The research was funded by a Technology Development Fund (TDF) from UR Ventures, a branch of the University of Rochester that helps transfer ideas and technologies from the Medical Center and the River Campus to the private sector for commercialization.

http://bit.ly/2jzxwhG

Scientists describe lab technique with potential to change medicine and research

Researchers who developed and tested a revolutionary laboratory technique that allows for the endless growth of normal and diseased cells in a laboratory are publicly sharing how the technique works.

WASHINGTON - The Georgetown University Medical Center (GUMC) researchers hope that by doing so, scientists around the world can realize the many of possibilities of "conditional reprogramming," which includes living biobanks, personalized and regenerative medicine, and novel cancer research.

Published in Nature Protocols, investigators demonstrate how conditional reprogramming (CR) works, and why it may be able to fill a number of clinical care and research voids.

CR is the only known system that can indefinitely grow healthy as well as cancer cells "as if they were just extracted from a patient, and expand them -- a million new cells can be grown in a week -- as long as needed," says the co-lead author Xuefeng Liu, MD, associate professor of pathology and a director in the Center for Cell Reprogramming at Georgetown University Medical Center.

No genetic modification is needed to coax the cells to grow -- all that is used are special "feeder" cells and a chemical inhibitor.

As one example, the researchers demonstrate they are able to use CR to produce new and healthy pancreatic beta islet cells that secrete insulin -- suggesting a promising avenue for type I diabetes research.

"A true cure for this kind of diabetes could be achieved by replacing the lost beta cells with new functional insulin producing cells," says Liu.

The researchers have also grown healthy and cancerous cells from airway tissues, retinas, prostates, breasts, and intestines, which replicate for extended periods with conditional reprogramming.

Since CR was developed and described by Liu, Richard Schlegel, MD, PhD, director of the Center for Cell Reprogramming, and their colleagues at Georgetown in 2011, scientists have been testing the ability of the cells to perform a number of advanced goals. The CR method has spread worldwide, for example, the National Cancer Institute cited the CR method in Precision Medicine Initiatives for oncology and drug discovery programs. Georgetown researchers have trained more than 100 scientists in the technique.

In the newly published protocol, the Georgetown researchers describe many other possibilities that CR offers: among them, living biobanks, personalized and regenerative medicine, and novel cancer research. For example, in a December study published in Oncotarget, Liu and Schlegel describe how CR allows them to grow both normal and primary cancerous prostate cells from a patient. This research represents a critical advance in the effort to understand the origin and drivers of this puzzling cancer.

Additionally, biobanking normal cells from a patient allows the possibility of using those cells in the future to infuse healthy cells into a damaged organ. "We can grow cells, freeze them, thaw them," Liu says. "Think about use of such cells for skin replacement, for organ patching, and cancer studies."

CR cancer cells also could allow oncologists to test and select a therapy based on an expanded laboratory population of a patient's individual cancer cells -- a procedure already conducted at Georgetown and published in the New England Journal of Medicine. An independent research study at Massachusetts General Hospital Cancer Center, published in Science, demonstrated that the CR method identified a combination of therapies for resistant lung cancer patients.

Several institutes have used CR platform for discovery of anti-cancer drug or new targets. For example, researchers at Helsinki established the first castration-resistant CR cells and discovered both known and novel drug sensitivities in prostate cancer cells, including navitoclax, which is currently being tested in clinical trials of castration-resistant prostate cancer. Yale scientists discovered Notch1 and SOX10 are potential new therapeutic targets of adenoid cystic carcinoma. Researchers at Fox Chase Cancer Center found that MYC-ERCC3 is new target for human pancreatic cancer and applied this novel target for drug discovery.

It may also be possible to fix damaged cells, using gene editing techniques, and then grow new, repaired cells to fix a wide variety of diseases, Liu says. "It is not unimaginable that we could take a tiny nose biopsy from a person with cystic fibrosis, correct the defect that causes the disease, then regrow the healthy cells to infuse back into the lung. Because the cells were derived from the patient, they would not be rejected."

Georgetown University has pending patent applications in US and internationally for conditional cell reprogramming and has been awarded a US patent by the United States Patent Office (9,279,106). This technology has been licensed exclusively to a company for further development and commercialization. Georgetown University and the inventors (Liu and Schlegel) receive payments and potential royalties from Propagenix. Schlegel is also a co-founder in the company that has a license to this technology.

Additional authors of the protocol include co-lead author Ewa Krawczyk, PhD, Frank A. Suprynowicz, Palechor-Ceron, DMD, Aleksandra Dakic, PhD, Vera Simic, Yun-Ling Zheng, MD, PhD, Praathibha Sripadhan, Chen Chen, Kie Lu, Tung-Wei Hou, Sujata Choudhury, PhD, Bhaskar Kallakury, MD, Anatoly Dritschilo, MD, Chris Albanese, PhD, and Seema Agarwal from Georgetown University Medical Center; Dean Tang, PhD, from the University of Texas MD Anderson Cancer Center; Thomas Darling, MD, PhD, and Rajesh Thangapazham, PhD, from the Uniformed Services University of the Health Sciences, Bethesda, Maryland; and Scott H. Randell, PhD, from the University of North Carolina School of Medicine.

Studies of conditional cell reprogramming were funded by the Center for Cell Reprogramming and by grants from the National Institutes of Health (R33CA177466, R21CA180524, and R01RR032315).

http://bit.ly/2joWxsD

Some Parts of Body Stay 'Alive' After Death, Evidence Suggests

Even after someone is declared dead, life continues in the body, suggests a surprising new study with important implications.

By Jen Viegas, Seeker | January 26, 2017 07:30am ET

Gene expression — when information stored in DNA is converted into instructions for making proteins or other molecules - actually increases in some cases after death, according to the new paper, which tracked postmortem activity and is published in the journal Open Biology.

"Not all cells are 'dead' when an organism dies," senior author Peter Noble of the University of Washington and Alabama State University told Seeker. "Different cell types have different life spans, generation times and resilience to extreme stress."

In fact, some cells seem to fight to live after the organism has died.

"It is likely that some cells remain alive and are attempting to repair themselves, specifically stem cells," Noble said.

Signs of Cellular Life

The international team of scientists, led by Alex Pozhitkov, studied zebrafish and mice and believe that the phenomenon occurs in all animals, including humans.

Gene transcription — the first step of gene expression, where a segment of DNA is copied into RNA — associated with stress, immunity, inflammation, cancer and other factors increased after death. And this could happen within hours or even days after the individual as a whole was declared dead.

Interestingly, gene transcription linked to embryonic development also increased. It's as though parts of the body essentially go back in time, exhibiting cellular characteristics of very early human development.

The Twilight of Death

The researchers identified a "step-wise shutdown" after death where some gene transcriptions diminished while others became more abundant. While the precise steps have yet to be defined, the scientists do not believe the process is random.

"Death is a time-dependent process," Noble remarked. "We have framed our discussion of death in reference to 'postmortem time' because on the one hand, there is no reason to suspect that minutes after an animal dies, gene transcription will abruptly stop."

"On the other hand," he added, "we know that within hours to days, the animal's body will eventually decompose by natural processes and gene transcription will end." The authors referred to the window of time between "death and the start of decomposition as the 'twilight of death' - when gene expression occurs, but not all of the cells are dead yet."

For years, researchers have noted that recipients of donor organs, such as livers, often exhibit increased risk of cancer following a transplant. The authors indicate there could be a link between "twilight of death" gene transcription and this increased cancer risk.

"It might be useful to prescreen transplant organs for increased cancer gene transcripts," Noble said, which might offer some insight on the health of the organ, though more research is needed.

If such a connection is established, the findings could help to explain why the donated organs of people who were young and healthy before death — for example, if they died in a sudden accident — could still lead to increased risk of cancer in the organ recipient.

Since gene transcription associated with cancer and inflammation also can increase postmortem, analyzing those activities and patterns could shed light on how these health problems arise in the living and how the body reacts once they have been established.

Ashim Malhotra, an assistant professor at Pacific University Oregon who was not involved with the study, said "one would expect genes involved in immunity and inflammation to [increase in response to a stimulus] right after... death because some cells remain alive for a short time and the transcriptional machinery is still operating in 'life mode.'"

Malhotra was nevertheless surprised that the process happened between 24 to 48 hours after death. The researchers concluded their investigations after that upper time limit, so the transcription could potentially go on for longer than two days.

Perhaps certain cells live longer than we think, but there could be another explanation that has not yet been considered.

Noble likens studying the dead to analyzing building collapses, in that both investigations can reveal what the original underlying structure was. "Like the twin towers on 9-11, we can get a lot of information on how a system collapses by studying the sequence of events as they unfold through time," he said. "In the case of the twin towers, we saw a systematic collapse of one floor at a time that affected the floors underneath it. This gives us an idea of the structural foundations supporting the building and we see a similar pattern in the shutdown of animals."

Putting Death on Hold

Malhotra hopes that the experiments of Noble, Pozhitkov and their team could be repeated with more sampling times—possibly going beyond 48 hours—in order to better understand the identified transcriptional dynamics. Since the new study is the first comprehensive investigation to assess changes in genetic transcription after organismal death, many questions remain.

Malhotra even raised the big question of raising the presumed dead. He wonders now if it might be possible to "put a hold on death" if the molecular processes underlying cellular death could be further determined and if scientists could develop specific ways to "interrupt the shutdown."

Arne Traulsen of the Max Planck Institute for Evolutionary Biology also expressed excitement over future related research. "I think this could be the start of a much more detailed analysis on how processes are being shut down after organismal death," Traulsen explained to Seeker.

"In spirit, death is probably more like turning a computer off and much less like turning a light bulb off," he added, referring to the computer-like step-by-step shutdown and intricacies involved. "We will see the consequences of this at some point, but I would not be surprised if this (new research) provides entirely new insights on the function of complex biological systems."

http://bit.ly/2jp1KAv

Pluto's Moon Charon Had Its Own, Icy Plate Tectonics

Surface cracks look like seafloor-spreading zones or rift valleys on Earth

By Jesse Emspak, SPACE.com on January 26, 2017

Pluto's largest moon, Charon, had a process like Earth's plate tectonics beneath its surface, driven by a freezing ice core that expanded and cracked the little world's crust, researchers said.

Using data from the New Horizons spacecraft, which flew past Pluto and Charon in July 2015, Ross Beyer and his colleagues on the mission team investigated features on Charon's surface to understand how they formed. They noted some similarities to what can be seen back home: Earth's geology is powered by huge plates of crust that float on a taffy-like mantle, crashing into one another. Certain features

on Charon seemed to have formed in the same way, the scientists found.

The researchers noticed there were fissures that looked a lot like seafloor-spreading zones or rift valleys on Earth. The scientists also saw depressed blocks of the surface surrounded by faults, called graben, and scarps, where one piece of land had moved vertically relative to the other.

Pluto's largest moon, Charon, is covered in cracks and fissures from when a liquid mantle froze and expanded, new research suggests. Credit: **NASA/JHUAPL/SwRI**

But the researchers didn't find any evidence of mountain building, a process on Earth caused by tectonic plates colliding.

“What we see are all of these fractures,” Beyer, a planetary scientist at NASA's Ames Research Center and a principal investigator at the SETI Institute, told Space.com. “On Earth, where you have plate tectonics, some plates move apart, but they must be colliding in other places, because the Earth is a sphere.”

Geological features like the Himalayas are direct illustrations of that collision, which happens because Earth's plates are moving apart in some other location, like the Atlantic Ocean or East African Rift, he said.; In contrast, “on Charon, we only saw the extensional features,” Beyer said. “As if the only thing we are seeing is pieces of crust moving away from each other.”

The planetary scientists said they think Charon once had a liquid mantle underneath a crust of mostly water ice. When the ice froze, it expanded, and Charon's crust had to stretch and crack to accommodate that expansion, they said. This doesn't happen with rock like Earth has; most of the time, melted rock will shrink when it becomes a solid.

“Chemically, Charon's surface is super bland, mostly water,” Beyer said. Data from New Horizons and earlier observations showed Charon's mean density. That density suggests it likely has a rocky core, or at least that there's some amount of rock in the little moon's makeup, but it would be a minority of the total mass.

Beyer noted that current models depict Charon forming as a result of a proto-Pluto colliding with something big and effectively splitting into two unequal pieces, one of which would become Charon. The initial collision would have generated some heat, and radioactive elements in Charon's core would have generated some more, he said. Initially the surface, made mostly of water with some ammonia, would freeze, but the layer between the rocky core and crust of ice would still be liquid, he added.

Amy Barr Mlinar, senior scientist at the Planetary Science Institute in Tucson, Arizona, told Space.com in an email that tidal forces could also contribute to keeping Charon's underground ocean liquid, though there aren't yet good models for that process in the Pluto-Charon system. And a lot of heat might not be necessary. “A little bit of ammonia (which has been detected on Charon’s surface, I believe) can significantly lower the freezing point of water,” she said. “This permits the satellite to have an ocean even if there isn’t much heat available — but Charon should have a decent amount of heat given its high rock fraction.”

Regardless of the heat source, Charon is small, with a lot of surface area relative to its volume. Objects with lots of surface area radiate heat away more quickly, which makes it more likely that the liquid mantle finally froze, probably within a few million years. One piece of evidence that the moon froze early is the cratered terrain; the number of craters shows that there hasn't been much geological activity for about 4 billion years at least, the study said.

When Charon's mantle froze, the moon's volume increased, stretching the crust. “So the solution is like a muffin getting bigger,” Beyer said. That process built the pattern of ridges and cracks that the scientists saw in the New Horizons images, he said. The team's research, currently in press, will appear in an upcoming issue of the journal Icarus.

http://bit.ly/2kG94qu

Twice-daily radiation therapy cuts deaths from head and neck cancer

Treating head and neck cancer patients with a twice-daily radiation therapy combined with chemotherapy could save more lives, according to new research presented at the European Cancer Congress 2017 [1].

Amsterdam, The Netherlands - The study, led by Dr Claire Petit, a resident in radiation oncology from Gustave Roussy cancer centre in Paris, included patients with tumours in their mouths, throats or voice boxes [2], that had already begun to spread to neighbouring tissue. These patients tend to have lower rates of survival than those whose cancer was diagnosed at an earlier stage [3].

The twice-daily treatment is known as hyperfractionated radiotherapy. By splitting the daily treatment in two portions, a higher and more effective dose can be given to patients. The researchers hope that this can be achieved without increasing side effects.

Around 600,000 people are diagnosed with head and neck cancer worldwide each year [4]. It can be difficult to treat because the area of the body affected contains so many vital structures, including those responsible for breathing, swallowing and speech.

The researchers used a relatively new technique called a network meta-analysis to bring together data from 117 different trials, including 28,804 patients from around the world. This allowed them to compare 16 different treatments to find out which was best at reducing the spread of cancer and deaths from the disease.

They discovered that the twice-daily treatment, when combined with chemotherapy, cut deaths by 20% compared to the best standard treatment of once-daily radiotherapy with chemotherapy. It also reduced the risk of the cancer getting worse by 23%.

Dr Petit told the Congress: "There are a number of new treatments that have shown promise in head and neck cancer trials. This large study has enabled us to compare several of these treatments to see which is best overall in terms of reducing mortality."

Dr Petit cautioned that she has not yet studied the side effects experienced by patients, either during treatment or in the longer term, and that more research is needed to examine this and to confirm the results. "Some of the studies we looked at did not include data on side effects; others did not follow patients long enough to pick up long-term side effects. This will be the focus of more research over the next year."

She added: "Moreover, the method we used, network meta-analysis, which combines direct and indirect treatment comparisons, is a new method that needs to be interpreted with prudence.

"However, this is an important finding for this group of patients who have a higher risk of their cancer recurring following treatment."

Professor Philip Poortmans, President-elect of ECCO and head of the Radiation Oncology Department at Radboud university medical center (Nijmegen, The Netherlands), said: "This research provides good evidence for the benefits of treating advanced stage head and neck cancer patients with a combination of twice-daily radiation therapy and chemotherapy, compared to one or even none of these separately. Before we can apply these very interesting results into daily clinical practice, we need to wait for the outcome of the next stage of this research - namely the evaluation of the short and long-term side effects. This is of utmost importance for the quality of life of the patients and their relatives.

"Moreover, it would be preferable to perform prospective trials to confirm these results. If that is not feasible, or if we cannot wait for

their outcome for some subgroups of patients who have the worst survival currently, then we should at least register carefully all the outcome parameters in prospective multi-centre databases so that they are available later on for analysis."

http://bit.ly/2kffRwt

Roots of Alzheimer's disease can extend as far back as the womb

Vitamin A deficiency could 'program' brain tissue

Biochemical reactions that cause Alzheimer's disease could begin in the womb or just after birth if the fetus or newborn does not get enough vitamin A, according to new research from the University of British Columbia.

These new findings, based on studies of genetically-engineered mice, also demonstrate that supplements given to newborns with low levels of vitamin A could be effective in slowing the degenerative brain disease.

"Our study clearly shows that marginal deficiency of vitamin A, even as early as in pregnancy, has a detrimental effect on brain development and has long-lasting effect that may facilitate Alzheimer's disease in later life," said Dr. Weihong Song, a professor of psychiatry and Canada Research Chair in Alzheimer's Disease.

For this research, Song built on previous studies that have linked low levels of vitamin A with cognitive impairments. In collaboration with Dr. Tingyu Li and others at Children's Hospital of Chongqing Medical University, they examined the effects of vitamin A deprivation in the womb and infancy on Alzheimer's model mice. These early developmental stages are crucial periods during which brain tissue is "programmed" for the rest of a person's life.

The researchers found that even a mild vitamin A deficiency increased the production of amyloid beta, the protein that forms plaques that smother and ultimately kill neurons in Alzheimer's disease. He also found that these mice, when deprived of vitamin A, performed worse as adults on a standard test of learning and memory.

Even when the mice deprived of vitamin A in the womb were given a normal diet as pups, they performed worse than mice who received a normal amount of the nutrient in the womb but were deprived after birth. In other words, the damage had already been done in the womb.

Still, Song and his collaborators also showed that some reversal is possible: Mice who were deprived in utero but then given supplements immediately after birth performed better on the tests than mice who weren't given such supplements.

"In some cases, providing supplements to the newborn Alzheimer's disease model mice could reduce the amyloid beta level and improve learning and memory deficits," said Song. "It's a matter of the earlier, the better."

The study, published today in Acta Neuropathologica, also included new evidence in humans of the vitamin A-dementia connection in later years. Examining 330 elderly people in Chongqing, Song and his collaborators found that 75 per cent of those with either mild or significant vitamin A deficiency had cognitive impairment, compared to 47 per cent of those with normal vitamin A levels.

However, Dr. Song cautions against overreacting to this news. Vitamin A deficiency, though common in many low-income regions of the world, is rare in North America, and excess intake of the nutrient could be harmful. Pregnant women in particular should not take excessive vitamin A supplements. A balanced diet is the best way to ensure adequate levels of the nutrient.

A portion of the research was funded by the National Natural Science Foundation of China and Canadian Institutes of Health Research.

http://bit.ly/2jhVC1P

Researchers list reasons not to lick a toad

New review of medicinal compounds in bufonidae secretions compiled

As human diseases become alarmingly antibiotic resistant, identification of new pharmaceuticals is critical. The cane toad and other members of the Bufonidae family produce substances widely

used in traditional folk medicine, but endangered family members, like Panama's golden frog, Atelopus zeteki, may disappear before revealing their secrets. Smithsonian scientists and colleagues at the University of Panama; Panama's government research center, INDICASAT AIP; Vanderbilt University in Tennessee; and Acharya Nagarjuna University in Guntur, India, created a compendium of the known chemicals produced by this amphibian family in the Journal of Ethnopharmacology, highlighting their largely unexplored potential for new drug discovery.

"We're slowly learning to breed several members of this amphibian family decimated by the chytrid fungal disease," said Roberto Ibáñez, staff scientist at the Smithsonian Tropical Research Institute (STRI) and in-country director of the Panama Amphibian Conservation and Rescue (PARC) project. "That's buying us time to study the chemicals they produce, but it's likely that animals in their natural habitats produce an even wider range of compounds."

Fifteen of 47 frog and toad species used in traditional medicine belong to the family Bufonidae. For millennia, secretions from their skin and from glands near their ears called parotid glands, as well as from their bones and muscle tissues have been used as remedies for infections, bites, cancer, heart disorders, hemorrhages, allergies, inflammation, pain and even AIDS.

The extensive review of the existing literature on toxins produced by this family revealed that two common Asian toad species, Bufo gargarizans and Duttaphrynus melanostictus, produce the anticancer remedies known as Chan Su and Senso. Another preparation used to treat cancer and hepatitis, Huachansu or Cinobufacini, is regulated by the Chinese State Food and Drug Administration. In Brazil, Rhinella schneideri intestines are applied to horses to treat the parasite Habronema muscae. In Spain, extract from the toad B. bufo is used to treat hoof rot in livestock. In China and North and South Korea, ranchers use the meat of B. gargarizans to treat rinderpest.

Only a small proportion of the more than 580 species in the Bufonidae family have been screened by scientists. "In Panama, not only do we have access to an amazing diversity of amphibian species, we will be able to use new mass spectrometry and nuclear magnetic resonance spectroscopy techniques to make it easier and cheaper to elucidate the chemical structures of the alkaloids, steroids, peptides and proteins produced by these animals," said coauthor Marcelino Gutiérrez, investigator at the Center for Biodiversity and Drug Discovery at INDICASAT AIP (Instituto de Investigaciones Cientificas y Servicios de Alta Tecnologia). "We're excited about learning more about the chemistry of this family."

It is thought that most of the chemicals produced in frog and toad skin protect them against predators. In the case of the genus, Atelopus, the majority of the toxins found in the skin are tetrodototoxins. In addition, zetekitoxins have been found in A. zeteki and chiriquitoxins in A. limosus, one of the first species that researches succeeded in breeding in captivity as well as in A. glyphus and A. chiriquiensis.

"Remarkably, toxins from a single frog skin can kill 130-1000 mice," said Candelario Rodriguez, researcher at INDICASAT and first author of the review. "The mechanism of action is to reduce cardiac rhythm, making these interesting candidates as therapeutic compounds. The golden frog, A. zeteki, one of Panama's national symbols, is the only species of the genus Atelopus that secretes zetekitoxins. Threatened by the chytrid fungal disease that infects its skin, as well as its collection for the exotic pet trade and by habitat destruction, if golden frogs were to disappear, they would take this potentially valuable chemical with them."

The chemical building blocks amphibians use to create toxic compounds come from sources including their diet, skin glands or symbiotic microorganisms. Toads in the genus Melanophryniscus sequester lipophilic alkaloids from their complex diet consisting of mites and ants. Preliminary studies showed that toxins found in a wild-caught species of Atelopus could not be isolated from frogs

raised in captivity: another reason to conserve frog habitat and to begin to explore the possibility of releasing frogs bred in captivity back into the wild.

More than 30 percent of amphibians in the world are in decline. Racing to stay ahead of the wave of disease spreading across Central America, Panama is leading the way in conservation efforts. The Smithsonian's PARC project identified several Atelopus species in danger of extinction. Researchers are learning how to create the conditions needed to breed them in captivity. Not only do animal caretakers at their facilities in Gamboa and El Valle, Panama, experiment to discover what the frogs eat, they also recreate the proper environment the entire frog life cycle: egg laying, egg hatching and tadpole survival, to successfully breed Atelopus. Each species has unique requirements making it an expensive challenge to create this Noah's ark for amphibians.

Rodríguez, C., Rollins-Smith, L., Ibáñez, R., Durant-Archibold, A., Gutiérrez, M. 2016. Toxins and pharmacologically active compounds from species of the family Bufonidae (Amphibia, Anura). Journal of Ethnopharmacology. doi:10.1016/j.jep.2016.12.021

http://bit.ly/2jJnFUq

TSRI scientists find brain hormone that triggers fat burning

Brain hormone that appears to trigger fat burning in the gut

LA JOLLA, CA - Biologists at The Scripps Research Institute (TSRI) have identified a brain hormone that appears to trigger fat burning in the gut. Their findings in animal models could have implications for future pharmaceutical development.

"This was basic science that unlocked an interesting mystery," said TSRI Assistant Professor Supriya Srinivasan, senior author of the new study, published today in the journal Nature Communications.

Previous studies had shown that the neurotransmitter serotonin can drive fat loss. Yet no one was sure exactly how. To answer that question, Srinivasan and her colleagues experimented with roundworms called C. elegans, which are often used as model organisms in biology. These worms have simpler metabolic systems than humans, but their brains produce many of the same signaling molecules, leading many researchers to believe that findings in C. elegans may be relevant for humans.

The researchers deleted genes in C. elegans to see if they could interrupt the path between brain serotonin and fat burning. By testing one gene after another, they hoped to find the gene without which fat burning wouldn't occur. This process of elimination led them to a gene that codes for a neuropeptide hormone they named FLP-7 (pronounced "flip 7").

Interestingly, they found that the mammalian version of FLP-7 (called Tachykinin) had been identified 80 years ago as a peptide that triggered muscle contractions when dribbled on pig intestines.

Scientists back then believed this was a hormone that connected the brain to the gut, but no one had linked the neuropeptide to fat metabolism in the time since.

The next step in the new study was to determine if FLP-7 was directly linked to serotonin levels in the brain. Study first author Lavinia Palamiuc, a TSRI research associate, spearheaded this effort by tagging FLP-7 with a fluorescent red protein so that it could be visualized in living animals, possible because the roundworm body is transparent. Her work revealed that FLP-7 was indeed secreted from neurons in the brain in response to elevated serotonin levels. FLP-7 then traveled through the circulatory system to start the fat burning process in the gut.

"That was a big moment for us," said Srinivasan. For the first time, researchers had found a brain hormone that specifically and selectively stimulates fat metabolism, without any effect on food intake.

Altogether, the newly discovered fat-burning pathway works like this: a neural circuit in the brain produces serotonin in response to sensory cues, such as food availability. This signals another set of neurons to begin producing FLP-7. FLP-7 then activates a receptor in intestinal cells, and the intestines begin turning fat into energy.

Next, the researchers investigated the consequences of manipulating FLP-7 levels. While increasing serotonin itself can have a broad impact on an animal's food intake, movement and reproductive behavior, the researchers found that increasing FLP-7 levels farther downstream didn't come with any obvious side effects. The worms continued to function normally while simply burning more fat.

Srinivasan said this finding could encourage future studies into how FLP-7 levels could be regulated without causing the side effects often experienced when manipulating overall serotonin levels.

In addition to Srinivasan and Palamiuc, authors of the study, "A tachykinin-like neuroendocrine signalling axis couples central serotonin action and nutrient sensing with peripheral lipid metabolism," were Tallie Noble of Mira Costa College and Emily Witham, Harkaranveer Ratanpal and Megan Vaughan of TSRI.

This study was supported by the National Institutes of Health's National Institute of Diabetes and Digestive and Kidney Diseases (grant R01 DK095804) and the NIH Office of Research Infrastructure Programs (grant P40 OD010440).

http://bit.ly/2jrcLS9

Yellow fever outbreak is killing off rare monkeys in Brazil

Rare monkeys in the forests of Brazil are being decimated by yellow fever.

By Adrian Barnett in São Paulo, Brazil

The outbreak started in late 2016 and, as is often the case in South America, it has spread to humans, killing at least 50 since the start of 2017. The authorities have rushed vaccines to hospitals, where long queues await inoculation.

But there is no vaccine for monkeys who are dying en masse in Espírito Santo and Minas Gerais, the two states so far worst hit.

“Some 80 to 90 per cent of the brown howler monkeys are infected or have already died,” says Sergio Mendes at the Federal University of Espírito Santo in Vitoria, Brazil. “This is a true catastrophe. These outbreaks happen periodically, but this is the worst I’ve ever seen.”

Mendes knows of 400 howler monkey deaths in the state, and he believes this is likely to be only 10 per cent of the total, with the greatest losses happening largely unseen in remote forested areas.

Atlantic titis and geoffroy’s marmosets found dead last week in Espirito Santo are also being tested for yellow fever. Both are unique to the Mata Atlantica, one of the world’s most species-rich and most-endangered tropical forests.

Other endemic primate species affected by the outbreak include the endangered buffy-headed marmoset and crested capuchin, and the critically endangered muriqui. There are only about 1000 muriqui individuals left in the wild, and their slow breeding time means numbers would take a long time to recover from yellow-fever deaths.

There are also unconfirmed reports of capuchin monkeys dying of suspected yellow fever in neighbouring Minas Gerais and in São Paulo states.

The virus is normally found in several forest-dwelling mammals, from marsupials to monkeys, and is transmitted by Haemagogus and Sabethes mosquitoes.

Marco Almeida, a veterinary epidemiologist from Rio Grande do Sul state’s health agency, says the current outbreak is unlikely to be caused by a new, more virulent form of yellow fever virus, as it is known to mutate very slowly.

Instead, he thinks recent prolonged and torrential rains provided ideal conditions for mosquitoes. Often delivering a week’s rain in a day, the deluges lasted over a month and may have weakened the monkeys by cutting the times when they can feed and challenging their immune systems.

“The mosquitoes can disperse across forest for up to 6 kilometres from their breeding point,” says Júlio-César Bicca-Marques, a primatolologist at Pontifical Catholic University of Rio Grande do Sul, “but they’ll also get rides in trucks. Plus, infected hunters, tree-cutters and agricultural workers can spread the virus.”

Yellow fever threat: A yellow fever epidemic has hit central Africa. Is Asia next?

It’s well known from lab tests that howlers are the most vulnerable to yellow fever of all of South America’s monkeys. “But with these

current high infection levels, the virus could spread to all of the region's 14 other primates," says Almeida.

"Part of the problem is forest fragmentation," says primate conservationist Karen Strier of University of Wisconsin-Madison. "Only 5 per cent of Mata Atlantica remains. So Mata Atlantica primate populations are small and isolated. Wipe one out, and natural recolonization is very difficult."

With monkeys being key seed-dispersers, the prognosis for both forest and primates is not good. Meanwhile, as the epidemic increases, ill-informed individuals have started attacking the region's monkeys, in the erroneous belief that they can spread yellow fever to humans directly.

http://bit.ly/2kG9Vxp

Corn turning French hamsters into deranged cannibals: research

The major consumption of corn leads to infanticide among the Great hamster

January 27, 2017 by Marlowe Hood

A diet of corn is turning wild hamsters in northeastern France into deranged cannibals that devour their offspring, alarmed researchers have reported. "There's clearly an imbalance," Gerard Baumgart, President of the Research Centre for Environmental Protection in Alsace, and an expert on the European hamster, told AFP on Friday.

"Our hamster habitat is collapsing." More common farther to the east, Cricetus cricetus in critically endangered in western Europe.

The findings, reported last week in the journal Proceedings of the Royal Society of London B, finger industrial-scale monoculture as the culprit. Once nourished by a variety of grains, roots and insects, the burrowing rodents live today in a semi-sterile and unbroken ocean of industrially grown maize, or corn.

The monotonous diet is leaving the animals starving, scientists discovered almost by accident. The problem is a lack of vitamins. In fact, one in particular: B3, or niacin.

Researchers led by Mathilde Tissier at the University of Strasbourg had set out to determine whether hamster diet affects their ability to reproduce in the wild.

Baby hamsters eaten alive

Earlier work had looked at the impact of pesticides and mechanised ploughing, which can destroy their underground homes, especially during hibernation in winter. But the possible link with what they eat remained unexplored.

A first set of lab experiments with wild specimens compared wheat and corn-based diets, with side dishes of clover or worms. There was virtually no difference in the number of pups born, or the basic nutritional value of the different menus. But when it came to survival rates, the difference was dramatic.

About four-fifths of the pups born of mothers feasting on wheat-and-clover or wheat-and-worms were weaned. Only five percent, however, of the baby hamsters whose mothers ate corn instead of wheat made it that far.

What was most disturbing is how they perished.

"Females stored their pups with their hoards of maize before eating them," the scientists reported. "Pups were still alive at that time."

The cannibal mothers showed other signs of abnormality. The usually cute-and-cuddly hamsters ran in circles, "climbing and pounding their feeders," when scientists entered the room. The females also had swollen and dark tongues, and blood so thick it was difficult to draw for samples. The researchers recognised the symptoms, and had a hunch as to what was causing them.

Vitamin B3 deficiency has been linked to 'black-tongue' syndrome in dogs, and a condition in humans called pellagra, also known as the "3-D" disease: diarrhoea, dementia and dermatitis, such as eczema.

"Improperly cooked maize-based diets have been associated with higher rates of homicide, suicide and cannibalism in humans," the researchers note.

'Dementia-like' behaviour

Pellagra is thought to have decimated some three million people in North America and Europe from the mid-18th to the mid-20th century. Tissier and her colleagues thought of a simple way to find out if their corn-crazed hamsters were suffering from a similar condition.

In a second set of experiments, they offered hamsters corn-based diets, one of them with B3 added. Sure enough, the vitamin-enriched diet was enough to eliminate the horrific symptoms, and prevent the female hamsters from eating their young.

The dire consequences of the B3-deficient corn diet, the scientists concluded, stemmed not from reduced maternal hormones, but rather a change in the nervous system that induced the same "dementia-like" behaviour diagnosed in humans.

"Knowing that these species already face many threats, and that most of them are in danger of extinction, it is urgent to restore a diverse range of plants in agriculture schemes," the researchers urged.

Baumgart, who has been fighting for years to protect the endangered rodents, agrees. "Monoculture in agriculture is really bad for biodiversity," he said. "Now we need to take concrete action."

More information: Mathilde L. Tissier et al. Diets derived from maize monoculture cause maternal infanticides in the endangered European hamster due to a vitamin B3 deficiency, Proceedings of the Royal Society B: Biological Sciences (2017). DOI: 10.1098/rspb.2016.2168

http://bit.ly/2kEP1LX

Diabetes or its rapid deterioration can be an early warning sign for pancreatic cancer

Onset of or rapid deterioration in existing diabetes could be a sign of early, hidden pancreatic cancer

Amsterdam, The Netherlands: Patients and their doctors should be aware that the onset of diabetes, or a rapid deterioration in existing diabetes that requires more aggressive treatment, could be a sign of early, hidden pancreatic cancer, according to research presented at the European Cancer Congress 2017 [1] today (Monday).

Ms Alice Koechlin, from the International Prevention Research Institute in Lyon, France, told the meeting that an analysis linking nearly a million patients with type 2 diabetes in Lombardy (Italy) and Belgium with recorded cases of pancreatic cancer showed that 50% of all pancreatic cancers cases in the two regions were diagnosed within one year of patients being diagnosed with type 2 diabetes and being given their first prescription to control it.

"In Belgium 25% of cases were diagnosed within 90 days and in Lombardy it was 18%. After the first year, the proportion of diagnosed pancreatic cancers dropped dramatically," she said. The researchers found that compared with patients who were able to continue with oral anti-diabetic drugs, patients in Belgium and in Lombardy had a 3.5-fold greater risk of being diagnosed with pancreatic cancer in the first three months after their first prescription for incretins (metabolic hormones that stimulate the pancreas to produce more insulin to lower blood glucose levels); this fell to a 2.3-fold risk in the next three to six months, to a two-fold risk for the next six to 12 months and 1.7-fold risk after the first year.

Among patients who already had type 2 diabetes and were managing it with oral anti-diabetic drugs, the switch to incretins or insulin happened faster among diabetic patients who were subsequently diagnosed with pancreatic cancer. In addition, a deterioration in their condition that necessitated them being switched to more aggressive anti-diabetic therapy with injections of insulin was associated with a seven-fold increased risk of being diagnosed with pancreatic cancer.

Ms Alice Koechlin, Professor Philippe Autier (also from the International Prevention Research Institute) and colleagues in Belgium and Italy used prescription data to identify 368,377 patients with type 2 diabetes in Belgium between 2008 and 2013 and 456,311 patients in Lombardy between 2008 and 2012. The data were linked to pancreatic cancer cases in the Belgian Cancer Registry and the

hospital discharge databases in Lombardy. There were 885 and 1,872 cases of pancreatic cancer diagnosed during this time in Belgium and Lombardy respectively.

Ms Koechlin Autier said: "Although it has been known for some time that there is an association between type 2 diabetes and pancreatic cancer, the relationship between the two conditions is complex. Incretin therapies reduce diabetic hyperglycemia through stimulating the release of insulin by the pancreas. These drugs are typically prescribed when the oral anti-diabetic drugs can no longer control blood glucose levels. Because of their stimulating effects on the pancreas, it has long been thought that the incretin therapies could promote the occurrence of pancreatic cancer. However, it is known that pancreatic cancer can cause diabetes. Our study shows that incretin therapies are often prescribed to patients whose diabetes is caused by a still undiagnosed pancreatic cancer. Because the pancreatic cancer finally becomes symptomatic and is thus diagnosed, it looks like it is the intake of incretin drugs that could be the trigger of the pancreatic cancer, while in reality, it is the pancreatic cancer that causes a deterioration of diabetes, which is followed by the prescription of incretins. This phenomenon is called 'reverse causation'. Our study also shows that the reverse causation observed for incretin drugs is also observed for other anti-diabetic therapies, in particular for insulin therapy.

"Doctors and their diabetic patients should be aware that the onset of diabetes or rapidly deteriorating diabetes could be the first sign of hidden pancreatic cancer, and steps should be taken to investigate it."

However, investigating whether or not a patient has undiagnosed pancreatic cancer is difficult, and the researchers say that using prescription databases in the way that they have could help to develop methods to identify which patients may have early, non-symptomatic pancreatic cancer.

"There is currently no good, non-invasive method for detecting pancreatic cancer that is not yet showing any visible signs or symptoms. We hope that our results will encourage the search for blood markers indicating the presence of pancreatic cancer, which could guide decisions to perform a confirmation examination like endoscopy," concluded Ms Koechlin.

Pancreatic cancer is one of the most lethal cancers, partly because it is difficult to detect at an early stage and because there are few effective treatments for it. Less than one per cent of people live for ten or more years after a diagnosis. In Europe around 104,000 new cases were diagnosed in 2012 and approximately the same number of people died from it. Worldwide there were an estimated 338,000 cases of pancreatic cancer diagnosed in 2012 and 330,000 people died from it.

Chair of the Congress and President of ECCO, Professor Peter Naredi, from the Sahlgrenska Academy, University of Gothenburg, Sweden, who was not involved with the research, commented: "Due to the severity of pancreatic cancer and because only a minority of cases are detected at a curable stage, we must find better ways for early detection. Some advances have been made in the search for blood biomarkers. The study by Autier and colleagues opens up the possibility to combine the diagnosis of an associated disease, type 2 diabetes, with blood biomarkers. It is a step in the right direction if we can increase the proportion of early diagnosed pancreatic cancers."

http://bit.ly/2kimHRG

Some early breast cancer patients benefit more from breast conservation than from mastectomy

BCT superior to mastectomy in certain types of breast cancer patients

Amsterdam, The Netherlands: Breast conserving therapy (BCT, breast conserving surgery combined with radiation therapy) is superior to mastectomy in certain types of breast cancer patients, according to results from the largest study to date, to be presented to the European Cancer Congress 2017 [1] today (Monday).

Professor Sabine Siesling, from the Netherlands Comprehensive Cancer Organisation (IKNL) and University of Twente and Mirelle

Lagendijk, MD, from the Department of Surgical Oncology, Erasmus MC Cancer Institute, Rotterdam, The Netherlands, and colleagues from other hospitals, studied survival nationwide in nearly 130,000 breast cancer patients, divided into two groups: those diagnosed between 1999-2005 and those diagnosed between 2006-2012. The patients selected from the Netherlands Cancer Registry had no metastases (spread of the cancer to organs other than the lymph nodes close to the tumour). To obtain information on cause of death, data were linked to the cause of death register.

Although randomised trials initiated in the 1980s have shown equal survival outcomes for BCT and mastectomy, trials often exclude elderly patients or patients with existing disease other than breast cancer (comorbidity). Studies with large, population-based groups, including comorbidity and those who are elderly, can add to the knowledge based on these trials and provide outcome that is more widely applicable and reflect daily practice. Several recent population-based studies showed a survival advantage for BCT. However, these studies tended to lack long-term follow-up, evaluated limited patient numbers, had differences in medication after surgery between both groups and lacked the data on cause of death that are needed to evaluate breast cancer-specific survival. All this could have led to the introduction of confounding factors such as severity of disease or death due to other causes, the researchers say.

In the current study, a number of prognostic factors such as age, stage, comorbidity, hormonal receptor and HER2 status [2], and differences in systemic treatments (medication after surgery) were included and considered as possible explanations for the previously reported survival differences between BCT and mastectomy. This enabled the identification of possible prognostic factors that might, in future, predict which patients could benefit most from BCT.

"We looked at two different groups in order to allow us to compare long-term outcomes in a more historical versus a more recent cohort, evaluating patients that had been able to benefit from more sophisticated diagnostics and therapies. A considerably superior survival, both specific to breast cancer and from any cause of death, was found for BCT in the early stage T1-2N0-1M0 [3] cancers in both time cohorts," says Prof Siesling.

To identify patients who could possibly benefit most from BCT, both time cohorts were divided into subgroups. Evaluation of T1-2N0-1M0 cancers, which are at a stage when metastasis to distant organs has not yet occurred, in both groups showed a considerable advantage for BCT in patients with increasing age, those with comorbidity, and those who did not receive chemotherapy.

"Although this study is based on retrospective data with much detailed data, and residual confounding factors cannot be ruled out completely, we believe that this information will have potential to greatly improve shared treatment decision-making for future breast cancer patients in those aged over 50 years and those with comorbidity," says Prof Siesling.

"However, we would like to emphasise that these results do not mean that mastectomy is a bad choice. For patients for whom radiotherapy is not suitable or feasible due to social circumstances, for whom the risk of late side effects of radiotherapy is high, or who have the prospect of a poor aesthetic outcome following BCT, a mastectomy may still be the preferable treatment option. Our study showed that BCT is at least as good as mastectomy and that some patients might benefit more than others from BCT in the future," Prof Siesling will conclude.

Abstract no: 4LBA, Practice Changing Trials III session, 12.00 hrs (CET) Monday 30 January, Room Veronesi.