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Two degrees of warming already baked in

Even if all emissions of greenhouse gases were stopped, Earth's temperature would rise about two degrees F

Even if humans could instantly turn off all our emissions of greenhouse gases, the Earth would continue to heat up about two more degrees Fahrenheit by the turn of the century, according to a sophisticated new analysis published in Nature Climate Change. And if current emissions continue for 15 years, odds are good that the planet will see nearly three degrees (1.5 C) of warming by then.

"This 'committed warming' is critical to understand because it can tell us and policy makers how long we have, at current emission rates, before the planet will warm to certain thresholds," said co-author Robert Pincus, a scientist with CIRES at the University of Colorado Boulder and NOAA's Physical Sciences Division. "The window of opportunity on a 1.5-degree [C] target is closing."

During United Nations meetings in Paris last year, 195 countries including the United States signed an agreement to keep global temperature rise less than 3.5 degrees F (2 C) above pre-industrial levels, and pursue efforts that would limit it further, to less than 3 degrees Fahrenheit (1.5 C) by 2100.

The new assessment by Pincus and lead author Thorsten Mauritsen, from the Max Planck Institute for Meteorology is unique in that it does not rely on computer model simulations, but rather on observations of the climate system to calculate Earth's climate commitment. Their work accounts for the capacity of oceans to absorb carbon, detailed data on the planet's energy imbalance, the climate-relevant behavior of fine particles in the atmosphere, and other factors. Among Pincus' and Mauritsen's findings:

Even if all fossil fuel emissions stopped in 2017, warming by 2100 is very likely to reach about 2.3 F (range: 1.6-4.1) or 1.3 degrees C (range: 0.9-2.3).

Oceans could reduce that figure a bit. Carbon naturally captured and stored in the deep ocean could cut committed warming by 0.4 degrees F (0.2 C).

There is some risk that warming this century cannot be kept to 1.5 degrees C beyond pre-industrial temperatures. In fact, there is a 13 percent chance we are already committed to 1.5-C warming by 2100.

"Our estimates are based on things that have already happened, things we can observe, and they point to the part of future warming that is already committed to by past emissions," said Mauritsen. "Future carbon dioxide emissions will then add extra warming on top of that commitment."

The research was funded by the Max-Planck-Gesellschaft, the U.S. Department of Energy and the National Science Foundation.

http://bit.ly/2vsXrhZ

'My kid is in there,' UT Health San Antonio imaging studies confirm

Structural and functional MRI in children resuscitated after drowning pinpoints site of anoxic brain injury

Highlights:

Structural and functional MRI in children resuscitated after drowning pinpoints the site of anoxic brain injury to regions controlling movement, while providing strong evidence that networks controlling perception and cognition remain largely intact.

In the not-too-distant future, it should be possible to target the area of injury with neuroprotective therapies -- now being tested in animal models -- when childhood drowning victims first arrive at the emergency room.

Children who are resuscitated after drowning can survive as prisoners inside their own bodies, awake but paralyzed. Drowning deprives the brain of oxygen, which can cause a form of anoxic brain injury (ABI). Unable to move or speak, 10 children with ABI studied at UT Health San Antonio exhibited variations of locked-in syndrome, a rare condition in adults and thought to be even rarer in children. This work

suggests that pediatric drowning may be one of the most common causes of locked-in syndrome.

Injury is localized, not diffuse

The scientists are from the Research Imaging Institute at The University of Texas Health Science Center, now called UT Health San Antonio. They studied brain images of the 10 children and reported July 31 in Human Brain Mapping that the pattern of ABI seen in childhood drowning is not widespread as was previously believed. Instead it is largely confined to a small but crucially important motor pathway supplied by a specific set of small arteries deep inside the brain.

Focal stroke

The injury is a focal stroke that, in the future, might be treated when children are first admitted to the hospital. That's if neuroprotective agents now being tested in animals are moved into human trials, said Peter T. Fox, M.D., professor in the Joe R. & Teresa Lozano Long School of Medicine at UT Health San Antonio and director of the Research Imaging Institute. Dr. Fox is senior author of the new paper and two other recent studies that report anatomical and functional effects on the brains of young children resuscitated after drowning.

Preservation of networks

"In the imaging studies we see preservation of visual, auditory, tactile and cognitive networks, and predictably, we also see damage to motor pathways and networks," Dr. Fox said.

The Research Imaging Institute developed a Neural Network-Based Behavioral Scoring System©, which is a score sheet for families and caregivers to submit their impressions of the awareness and cognition that their children display. Some of the children are able to communicate via eye movements. Others are not able to do so.

Family impressions

"In locked-in syndrome, the patients' families are typically the first to report return of consciousness," Dr. Fox said. "They are around their children all the time and they remember the child's personalities and preferences from before the injury."

The brain system that's most associated with awareness is called the default-mode network. The team found a strong correlation between social behaviors and preservation of this network, Dr. Fox said.

Machinery for awareness

"Even in the most severely affected of the children, we have evidence that at least the machinery for awareness of themselves and others remains functioning," he said.

"Does it mean that they're fully conscious? Well, we can see that the machinery is still intact."

Relief to parents

The finding is exciting and a relief to parents like Liz Tullis, whose son, Conrad, suffered ABI from drowning and was resuscitated. She founded the Conrad Smiles Fund, which has supported the research, and is a co-author on the three papers.

"Some parents, like Liz, are quite convinced that their kid is in there, thinking," Dr. Fox said. "Perhaps clinicians are telling them that their child is not aware, and they want another opinion. They came to be a part of the study for another opinion."

In future, treatment in hospital envisioned

If this new observation -- that ABI from childhood drowning can cause a focal stroke in the motor pathways -- becomes widely known among clinicians, and if treatments that are effective in animal models can be translated to humans, children could be treated immediately after hospital admission and much function could be saved, Dr. Fox said. "This is a new syndrome," he said. "It's not taught in medical school. This is all new neuroscience."

Hope for parents

"When Conrad survived his accident, I was not given much hope or guidance; in fact I was encouraged to institutionalize Conrad," Tullis said. "Other families were encouraged to withdraw care. Because ABI is believed to be 'generalized' brain damage, the prevailing medical

prognosis is grim and any treatment or recovery is considered too difficult if not impossible.

"The results of this study are groundbreaking," she continued. "Simply publicizing the results of Dr. Fox's research will have a huge impact on families. The fact that these children are 'in there' has never been communicated to the medical community and will improve the support we receive. The fact that this study can lead to more advancements in the understanding and treatment of ABI provides additional hope to fuel the fire of our love and dedication to our children."

The team has published its research findings in clinical journals so that this will have an impact on care delivery, Dr. Fox said.

Maximizing intellectual stimulation

Thirteen years after his accident, Conrad Tullis is 15 and a high school sophomore in San Antonio. Parents like Liz Tullis provide "environmental enrichment" opportunities for their locked-in children.

"If you believe that your kid is aware, then you want him to be stimulated, and have the opportunity to develop his own knowledge base and develop his own personality and intellect," Dr. Fox said. "This research can guide care toward brain systems that are preserved, and it can encourage families to provide an enriched environment."

Acknowledgments

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http://bit.ly/2wllD2j

Astronomers find that the sun's core rotates 4 times faster than its surface

Surprising observation might reveal what the sun was like when it formed

The sun's core rotates nearly four times faster than the sun's surface, according to new findings by an international team of astronomers. Scientists had assumed the core was rotating like a merry-go-round at about the same speed as the surface.

"The most likely explanation is that this core rotation is left over from the period when the sun formed, some 4.6 billion years ago," said Roger Ulrich, a UCLA professor emeritus of astronomy, who has studied the sun's interior for more than 40 years and co-author of the study that was published today in the journal Astronomy and Astrophysics. "It's a surprise, and exciting to think we might have uncovered a relic of what the sun was like when it first formed."

The rotation of the solar core may give a clue to how the sun formed. After the sun formed, the solar wind likely slowed the rotation of the outer part of the sun, he said. The rotation might also impact sunspots, which also rotate, Ulrich said. Sunspots can be enormous; a single sunspot can even be larger than the Earth.

The researchers studied surface acoustic waves in the sun's atmosphere, some of which penetrate to the sun's core, where they interact with gravity waves that have a sloshing motion similar to how water would move in a half-filled tanker truck driving on a curvy mountain road. From those observations, they detected the sloshing motions of the solar core. By carefully measuring the acoustic waves, the researchers precisely determined the time it takes an acoustic wave to travel from the surface to the center of the sun and back again. That travel time turns out to be influenced a slight amount by the sloshing motion of the gravity waves, Ulrich said.

The researchers identified the sloshing motion and made the calculations using 16 years of observations from an instrument called

GOLF (Global Oscillations at Low Frequency) on a spacecraft called SoHO (the Solar and Heliospheric Observatory) -- a joint project of the European Space Agency and NASA. The method was developed by the researchers, led by astronomer Eric Fossat of the Observatoire de la Côte d'Azur in Nice, France. Patrick Boumier with France's Institut d'Astrophysique Spatiale is GOLF's principal investigator and a co-author of the study.

The idea that the solar core could be rotating more rapidly than the surface has been considered for more than 20 years, but has never before been measured.

The core of the sun differs from its surface in another way as well. The core has a temperature of approximately 29 million degrees Fahrenheit, which is 15.7 million Kelvin. The sun's surface is "only" about 30,000 degrees Fahrenheit, or 5,800 Kelvin.

Ulrich worked with the GOLF science team, analyzing and interpreting the data for 15 years. Ulrich received funding from NASA for his research. The GOLF instrument was funded primarily by the European Space Agency.

SoHO was launched on Dec. 2, 1995 to study the sun from its core to the outer corona and the solar wind; the spacecraft continues to operate.

http://bit.ly/2uVTa5c

Pig-hunting dogs and humans are at risk of a disease that can cause miscarriages and infertility

A disease called swine brucellosis is emerging in New South Wales, carried by feral pigs.

July 31, 2017 by Richard Malik And Siobhan Mor, The Conversation

Endemic to feral pigs in Queensland, and sometimes infecting the dogs used to hunt them, it can be transmitted to humans through blood contact with infected pigs. A number of people have already been infected in NSW.

Recreational pig hunting in rural Australia is a widespread control method for the roughly 24 million feral pigs who call Australia home.

The ethics of this undertaking is open to debate – many authorities consider poisoning more efficient and more ethical than hunting. But regardless of this controversy, the emergence of swine brucellosis illustrates the risk that comes with hunting.

Feral pigs are found in every state and territory in Australia. **Shutterstock**

How swine brucellosis spreads

Hunting and killing feral pigs is risky to all participants. Adult pigs are large, powerful animals, and their tusks can inflict serious injuries to both human and canine combatants. Despite the leather armour given to pig-hunting dogs, they commonly receive penetrating injuries. These can cause substantial wounds, peritonitis (inflammation of the lining of the abdominal cavity) and even death.

These risks are of course well understood by the people that hunt pigs. But regrettably, many tend to their dogs' injuries without veterinary assistance. Most feral pigs show little outward signs of the disease, so the danger to man and dog can be hidden even to an experienced "pigger".

People and dogs can be infected if they have a break in their skin (such as a minor wound or abrasion) that becomes contaminated with the blood or tissue of a pig or dog carrying the pathogen. This can occur during capture, or when the pig carcass is "dressed" in the field. Veterinarians in Australia have also been infected with *Brucella suis* following surgery on infected dogs.

In people, brucellosis is a systemic disease which can result in undulant fever, lassitude, sore joints and back pain. More serious cases involve orchitis and epididymitis (swollen testicles), miscarriage as well as kidney, liver or cardiovascular disease. As TIME magazine famously reported in 1943, "the disease rarely kills anybody but it often makes a patient wish he were dead."

Brucellosis (caused by bacterium *Brucella suis*) can usually be rapidly diagnosed through blood tests and other clinical investigations, as long as the history of pig hunting is disclosed to the medical team. Although there is usually a connection to pig hunting, humans can also be infected by accidental contact with the organism in diagnostic laboratories.

Swine brucellosis is seen only in feral pigs in Australia, and there is currently no risk to humans from pigs kept in modern intensive piggeries. The disease is considered endemic in Queensland, but it appears to be emerging in NSW. We see it more and more commonly in canine patients in the northern parts of the state, as the disease extends its biological range.

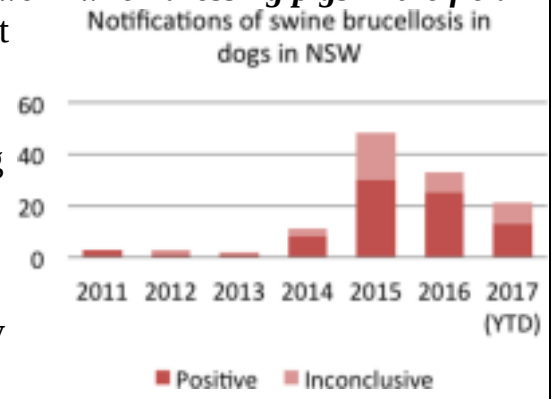
This might be a natural process, although some people suspect the deliberate (and illegal) capture and relocation of young feral pigs from Queensland to NSW plays a key role in the spread of infection.

How to protect yourself and your animals

There are some simple recommendations which will reduce the risk of infections in people who hunt pigs, and their families:

- ***when handling pig carcasses, always cover any skin cuts with waterproof dressings, and if possible, use disposable gloves***
- ***minimise exposure to blood, fluids and organs and always wash hands and arms with soap and water afterwards***
- ***mesh protective gloves should be worn when dressing pigs in the field***

Our focus has been the disease that occurs in "pig dogs", who are at risk of infection from hunting injuries and the practice of feeding raw feral pig meat or offal to dogs after they are dressed in the field. Non-hunting "house dogs" of pig hunters can also be infected if they are fed feral pig flesh.



There's been a dramatic increase in swine brucellosis cases in NSW. **Data from the Department of Primary Industries**

This can make the diagnosis harder, as the relationship with pig hunting is not apparent.

To make matters even more complex the disease can have a long incubation period, so dogs from the country can be infected while young, make their way into pounds and be rescued by people from urban areas where pig hunting is alien, and not often considered by city veterinarians. In one case, a female dog in Sydney had two years of extensive investigation into her lameness and back pain before diagnosis.

Dogs with swine brucellosis can develop various signs including swollen testicles, back pain, joint involvement, abortion as well as the less specific signs of fever and lassitude. The NSW Department of Primary Industries currently provides free testing through the Elizabeth Macarthur Agriculture Institute in New South Wales.

It's not all bad news. While euthanasia may be recommended to protect public health, preliminary evidence suggests the disease can be treated in dogs using combination therapy with two antibiotics (rifampicin and doxycycline) which are relatively inexpensive.

Ideally, this is combined with castration or removal of the ovaries and uterus, to remove any residual infected gonadal tissue. It's too early to tell whether dogs are cured for good but the results are looking promising.

Prevention is always better than cure, so one obvious solution would be to use poisoning of feral pigs as a method of population control rather than hunting.

If hunting cannot be prevented, it is strongly recommended that feral pig meat is thoroughly cooked before feeding it to dogs or people – this also kills the parasite that cause sparganosis and the bacteria which cause Q fever. Do not let pig-hunting dogs lick humans, and always wash your hands after contact with feral pigs or dogs.

More information about brucellosis and feral pig hunting and brucellosis in dogs can be found on government websites.

http://bbc.in/2vsXZ7I

Better drugs, faster: The potential of AI-powered humans

Scientists working in tandem with artificial intelligence (AI) could slash the time it takes to develop new drugs - and, crucially, the cost - say tech companies.

By Emma Woollacott Technology of Business reporter

Developing pharmaceutical drugs is a very expensive and time-consuming business. And as AstraZeneca found out last week, disappointing drug trials can knock millions off your stock market value in a flash.

So the faster we can identify promising molecules that could be turned into viable drugs, the better.

This is why pharmaceutical companies, such as GlaxoSmithKline (GSK), Merck, Sanofi and Johnson & Johnson, are now turning to artificial intelligence (AI) to help them.

Prof Andrew Hopkins is chief executive of Exscientia, an AI-based drug discovery firm that has recently signed a £33m deal with GSK.

He claims that AI and human beings working together in so-called "centaur teams" can help identify candidate molecules in a quarter of the usual time and at a quarter of the cost.

In Greek mythology, the centaur was half human, half horse - and very powerful and fast as a result. AI is giving scientists such extra powers, Prof Hopkins believes.

Successful drug discovery relies on precise understanding of how a disease affects our biological systems, says Pamela Spence, global life sciences industry leader at consultancy firm EY.

"Once that is known, scientists then search for molecules that can selectively interact with this 'target' and reverse that disruption or slow its impact - a 'hit' molecule," she explains.

Scientists often talk of a disease as the target and the molecule as a weapon being fired at it.

But this process of drug discovery - traditionally carried out by small teams of scientists painstakingly testing each potential target and hit molecule in the hope of finding a winner - is an enormously time-consuming approach that also has a very high failure rate.

So bringing in AI is like having a research assistant who can solve problems by systematic and relentless search at incredible speeds, she says.

"What might work - and equally importantly what might not work - can be identified first by the AI supercomputer 'in silico'," she says.

This is the medical term for research carried out by computer, as opposed to "in vitro" - think test tubes - and "in vivo" - testing on animals and humans.

As carrying out human clinical trials accounts for the vast bulk of drug discovery cost, the sooner we can identify when something isn't going to work, the less money will be wasted.

"Then the physical testing can be done on a smaller number of potential new medicines... and a much higher success rate can be achieved," says Ms Spence.

Exscientia's AI algorithm crunches masses of data, from the structure of diseases to the efficacy of existing drugs, from peer-reviewed studies to observations of slides under a microscope.

And all these possibilities are narrowed down in a process Prof Hopkins likens to natural selection.

"We're not trying to rule out the uncertainty - this is messy, dirty data," he says. "There are very interesting analogies between how human creativity works and evolution."

The aim is to come up with small molecules as candidates for up to 10 disease-related targets that can then be put through clinical tests.

"Every pill you make might cost pence to manufacture, but it's actually a precision-engineered product," says Prof Hopkins, who is also chair of medicinal informatics at the UK's Dundee University.

"There's an almost infinite number of other molecules it could have been. You have to make decisions as to what one might be safe and efficacious," he says. "Most don't lead to anything."

This AI-driven approach also makes it easier to come up with molecules that can have two distinct targets.

For example, a cancer drug could also improve the immune system as well as tackle the disease.

GSK is getting behind the idea and has recently set up a discovery performance unit focused on enhancing drug discovery through the use of "in silico" technology - including AI, machine learning and deep learning.

The drive is being led by John Baldoni, GSK's head of R&D.

"We have a number of these deals that we are putting in place; the one with Exscientia is probably the one that's furthest along, but we have a few others in flow and a few internal projects ourselves," he says.

"The cost of discovery from target to launch is roughly $1.7bn [£1.3bn]. The cost of what we're talking about here, from target to clinic, is about 33% of that, and it takes about five-and-a-half years.

"Our goal is to reduce that to one year, and reduce the cost commensurate with that." AI is also finding its way into other aspects of the drug discovery process.

Benevolent AI, for example, uses natural language processing to sift through published literature, such as chemical libraries, medical databases and scientific papers, to draw conclusions about possible new drug candidates.

Earlier this year, one of its candidates for a drug to treat motor neurone disease - also known as ALS (Amyotrophic Lateral Sclerosis) - was found to prevent the death of motor neurones in cells taken from real patients, and delayed the onset of the disease in animals.

"We are incredibly encouraged by these findings," says Benevolent AI founder and chairman Ken Mulvaney.

Patients should be encouraged, too. AI-based drug discovery promises to bring more effective, cheaper drugs on to the market much more quickly.

http://bit.ly/2voj42f

Signs of Alzheimer's found in chimpanzees for the first time

We may not be alone in our struggle against Alzheimer's disease.

By Helen Thomson

For the first time, the plaques and tangles that characterise the condition have been found in the brains of elderly chimpanzees, although it is unclear if they cause dementia in the animals.

In the brains of people with Alzheimer's, a protein called beta-amyloid accumulates and forms sticky plaques between brain cells. These plaques trigger changes in another protein called tau, causing it to form tangles. Together, these plaques and tangles are thought to kill brain cells, leading to dementia.

It is difficult to study the disease and develop treatments for it because other species seem not to develop plaques and tangles. The only time they've both been seen in another animal's brain was in a 41-year-old chimpanzee, but they were thought to be the result of a stroke.

But Melissa Edler, now at Northeast Ohio Medical University, and her colleagues have had the rare chance to study 20 brains from older chimpanzees, aged between 37 and 62. The team examined four areas of the chimps' neocortex and hippocampus – brain regions most commonly affected by Alzheimer's in humans. They discovered beta-amyloid plaques and early forms of tau tangles coexisting in 12 of the chimp brains and, as in humans, they saw increasingly larger volumes of plaques in the chimp brains of more advanced age (Neurobiology of Aging, DOI: 10.1016/j.neurobiolaging.2017.07.006).

It is not clear whether these plaques and tangles lead to the same kind of cognitive decline in chimps that we experience. "Our samples had been collected over decades, without any consistent or rigorous cognitive data accompanying them," says Mary Ann Raghanti of Kent State University, Ohio, in whose lab the work was performed. "So it wasn't possible to say whether the chimps had devastating cognitive loss or not."

However, so far, there are no examples of chimps with Alzheimer's-like dementia. "I'm cautious to say that they don't get this kind of devastating decline, but we haven't seen it yet," says Raghanti.

This study contributes to growing evidence that the classic plaques and tangles of Alzheimer's disease may be by-products rather than the cause of the disease, says Gary Kennedy, director of the Division of Geriatric Psychiatry at Albert Einstein College of Medicine in New York. "That great apes demonstrate the pathology but not the precipitous decline of dementia reinforces this notion."

Humans may have something unique about their brain that predisposes us to the cognitive decline that accompanies plaques and tangles, says Raghanti. "If we can identify those differences between the human and chimp brain then we might be able to pinpoint what is mediating the degeneration. That could be a target for drug treatment."

http://bit.ly/2voj42f

Finally, a scientific test can prove chronic fatigue syndrome

Protein concentrations in the bloodstream clearly show CFS, or myalgic encephalomyelitis, is an inflammatory disease.

It has been described variously as a chronic disease, a psychiatric disorder and a figment of the imagination but now research has finally succeeded in identifying biomarkers that confirm and quantify the existence of chronic fatigue syndrome (CFS).

CFS, or myalgic encephalomyelitis, is characterised by persistent fatigue over a long period, which significantly impacts quality of life. The fatigue is unrelated to levels of physical activity. Medication and therapy have little effect.

What causes CFS is still unknown. In many cases symptoms begin suddenly, typically after a short flu-like illness. In others the onset is gradual, over a period of weeks or months. Three-quarters of sufferers are women.

The lack of defined causal agents has often made an already unpleasant situation even worse for sufferers, with some doctors, employers and insurers refusing to believe CFS is anything more than an imagined disease.

The absence of unequivocal physical criteria also means the condition can be over-diagnosed. A study led by Natalia Palacios of the University of Massachusetts found that 80% of people diagnosed with CFS did not have the necessary symptoms described on the checklist drawn up by the US Centers for Disease Control and Prevention.

Now researchers at Stanford University in California have identified 17 proteins involved in immune system signalling that signify the presence of the condition. The concentration of the proteins, or cytokines, in the bloodstream correlate exactly with the severity of symptoms.

In a paper published in the Proceedings of the National Academy of Sciences, the scientists say the presence of elevated levels of the proteins show inflammation is a key driver for CFS.

Having definite and quantifiable markers for the condition will go a long way towards CFS being recognised as a bona fide illness, despite the continuing mystery surrounding its cause. "I have seen the horrors of this disease, multiplied by hundreds of patients," says lead researcher Jose Montoya. "It has been observed and talked about for 35 years now, sometimes with the onus of being described as a psychological condition. But chronic fatigue syndrome is by no means a figment of the imagination. This is real."

Apart from tiredness, CFS symptoms differ substantially from person to person, ranging from mental impairment to digestive problems to muscle pain. This variety often confounds diagnosis, and in the past has added to suggestions myalgic encephalomyelitis is at best a misidentification of other disorders rather than a disease in its own right.

Montoya's colleague Mark Davis says the identification of the proteins means a proper test that records inflammation levels rather than external symptoms may soon be possible.

"There has been a great deal of controversy and confusion surrounding ME/CFS – even whether it is an actual disease," he says. "Our findings show clearly that it's an inflammatory disease and provide a solid basis for a diagnostic blood test."

http://bit.ly/2vtd6Ol

For white middle class, moderate drinking is linked to cognitive health in old age

More likely to live to the age of 85 without dementia or other cognitive impairments than non-drinkers

Older adults who consume alcohol moderately on a regular basis are more likely to live to the age of 85 without dementia or other cognitive impairments than non-drinkers, according to a University of California San Diego School of Medicine-led study.

The findings are published in the August issue of the Journal of Alzheimer's Disease.

Previous studies have found a correlation between moderate alcohol intake and longevity. "This study is unique because we considered men and women's cognitive health at late age and found that alcohol consumption is not only associated with reduced mortality, but with greater chances of remaining cognitively healthy into older age," said senior author Linda McEvoy, PhD, an associate professor at UC San Diego School of Medicine.

In particular, the researchers found that among men and women 85 and older, individuals who consumed "moderate to heavy" amounts of alcohol five to seven days a week were twice as likely to be cognitively healthy than non-drinkers. Cognitive health was assessed every four years over the course of the 29-year study, using a standard dementia screening test known as the Mini Mental State Examination.

Drinking was categorized as moderate, heavy or excessive using gender and age-specific guidelines established by the National Institute on Alcohol Abuse and Alcoholism. By its definition, moderate drinking involves consuming up to one alcoholic beverage a day for adult women of any age and men aged 65 and older; and up to two drinks a day for adult men under age 65. Heavy drinking is defined as up to three alcoholic beverages per day for women of any adult age and men 65 and older; and four drinks a day for adult men under 65. Drinking more than these amounts is categorized as excessive.

"It is important to point out that there were very few individuals in our study who drank to excess, so our study does not show how excessive or binge-type drinking may affect longevity and cognitive health in aging," McEvoy said. Long-term excessive alcohol intake is known to cause alcohol-related dementia.

The researchers said the study does not suggest drinking is responsible for increased longevity and cognitive health. Alcohol consumption, particularly of wine, is associated with higher incomes and education levels, which in turn are associated with lower rates of smoking, lower rates of mental illness and better access to health care.

The UC San Diego School of Medicine research team adjusted the statistical analyses to remove confounding variables, such as smoking or obesity, but noted the study is based only on statistical relationships between different demographic factors, behaviors and health outcomes. There remain on-going debates about whether and how alcohol impacts lifespan or potentially protects against cognitive impairments with age.

One of the study's advantages, however, is that the data derive from a relatively homogenous population in a geographically well-defined area. All of the 1,344 older adults (728 women; 616 men) who participated in the study are from Rancho Bernardo, a white-collar, middle-to-upper-middle-class suburb in San Diego County. More than 99 percent of the study participants, tracked from 1984 to 2013, are Caucasian with at least some college education.

"This study shows that moderate drinking may be part of a healthy lifestyle to maintain cognitive fitness in aging," said lead author Erin Richard, a graduate student in the Joint San Diego State University/UC San Diego Doctoral Program in Public Health.

"However, it is not a recommendation for everyone to drink. Some people have health problems that are made worse by alcohol, and others cannot limit their drinking to only a glass or two per day. For these people, drinking can have negative consequences."

Co-authors include: Donna Kritz-Silverstein, Gail A. Laughlin, and Elizabeth Barrett-Connor, UC San Diego; and Teresa T. Fung, Simmons College and Harvard University.

http://bit.ly/2uW6Pte

Revealed: brain 'switch' tells body to burn fat after a meal

Discovery of a mechanism by which the brain coordinates feeding with energy expenditure

Scientists at Monash University's Biomedicine Discovery Institute have found a mechanism by which the brain coordinates feeding with energy expenditure, solving a puzzle that has previously eluded researchers and offering a potential novel target for the treatment of obesity.

Obesity - a major risk factor for many diseases including cardiovascular disease, Type 2 diabetes, liver disease and several cancers - is at epidemic levels in Australia.

Researchers from the Metabolic Disease and Obesity Program have shown in laboratory models that feeding controls the 'browning' of fat, that is, the conversion of white fat, which stores energy, into brown fat, which expends it. Fat in the human body is stored in specialised cells called adipocytes, which can change from white to brown states and back again.

Their study, published in Cell Metabolism today, shows that after a meal the brain responds to circulating insulin, which is increased after a rise in blood glucose. The brain then sends signals to promote the browning of fat to expend energy. By contrast, after a fast, the brain instructs these browned adipocytes to once more convert into white adipocytes, storing energy. These processes help prevent both excess weight gain and excess weight loss in response to feeding and fasting, meaning body weight remains relatively stable over time.

The researchers showed that the brain's ability to sense insulin and coordinate feeding with energy expenditure via browning is controlled by a switch-like mechanism turned on after fasting to inhibit the response to insulin, repressing browning and conserving energy, and turned off after feeding to facilitate the insulin response to promote browning and to expend energy.

"What happens in the context of obesity is that the switch stays on all the time - it doesn't turn on off during feeding," lead researcher Professor Tony Tiganis said.

"As a consequence, browning is turned off all the time and energy expenditure is decreased all the time, so when you eat, you don't see a commensurate increase in energy expenditure - and that promotes weight gain," Professor Tiganis said.

Previous investigations by the researchers that showed how the brain coordinates white adipose tissue browning attracted considerable attention after it was published in early 2015.

"For a long time, the missing piece to the puzzle was always why this occurs in the body," first author Dr Garron Dodd said.

"We've shown not only why this occurs but also the fundamental mechanism involved. It's very exciting," Dr Dodd said.

The researchers are further exploring the possibility of inhibiting the switch for therapeutic purposes to promote the shedding of excess fat.

"Obesity is a major and leading factor in overall disease burden worldwide and is poised, for the first time in modern history, to lead to falls in overall life expectancy," Professor Tiganis said.

"What our studies have shown is that there is a fundamental mechanism at play that normally ensures that energy expenditure is matched with energy intake. When this is defective, you put on more weight. Potentially we may be able to rewire this mechanism to promote energy expenditure and weight loss in obese individuals. But any potential therapy is a long way off," he said.

Researchers from Germany collaborated on the study.

This research was supported by the Australian National Health and Medical Research Council.

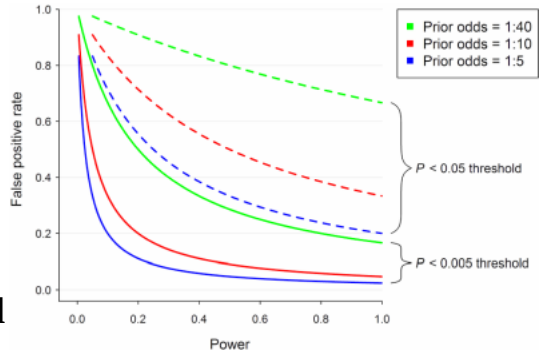
http://bit.ly/2vlYr7r

Redefine statistical significance: Large group of scientists, statisticians argue for changing p-value from .05 to .005

Arguments for changing the p-value from .05 to .005

August 1, 2017 by Bob Yirka report

Phys.org - A large group of scientists and statisticians has uploaded a paper to the PsyArXiv preprint server arguing for changing the p-value from .05 to .005. The paper outlines their reasons for suggesting that the commonly used value for assigning significance to results be changed.



Relationship between the P-value threshold, power, and the false positive rate. **PsyArXiv, 22 July 2017**

Some science is cut and dried: If you drop a ball from a tower, for example, it will fall to the ground under normal circumstances. Unfortunately, a lot of other science is not nearly so definitive—the science of investigating, producing and using pharmaceuticals, for example. Not all drugs work as expected in all people under all conditions. Uncertainty is prevalent in many areas, including astronomy, physics and economics. Because of this, the scientific community has settled on a means for obtaining the p-value that offers a measure of an experiment's success. Different p-values mean different things, of course, but the most prominent is the one that represents what has come to be known as statistical significance, which has historically been set at .05. But now, this new paper suggests that the bar has been set too low, and is therefore contributing to the problem of irreproducible findings in research efforts.

One of the main problems with the p-value, some in the statistics field have suggested, is that non-statisticians do not really understand it and because of that, use it incorrectly. It cannot be used, for example, to declare that a new drug has a 95 percent chance of working if it is used in the prescribed way. It is also not a way of interpreting how true something is, they note. Instead, it is defined as the probability of an outcome when conducting a test that is equal to or "more extreme" than the result if the null hypothesis (nothing happened) is true.

But even when it is used correctly, it does not offer a strong enough measure of evidence, according to the authors. Thus, they suggest changing the p-value to .005. They claim doing so would reduce the rate of false positives from the current 33 percent down to 5 percent.

More information: Benjamin, Daniel J et al. "Redefine Statistical Significance". PsyArXiv, 22 July 2017. Web. dx.doi.org/10.17605/OSF.IO/MKY9J

http://bbc.in/2vlEoFU

Did the first flower look like this?

All living flowers ultimately derive from a single ancestor that lived about 140 million years ago, a study suggests.

By Prof Sarah Gabbott Science writer

Scientists combined models of flower evolution with the largest data set of features from living flowers ever assembled. From this the team was able to infer the appearance of the ancestral flower. The flower had many concentric cycles of petal-like organs in sets of three, arranged in whorls, and was bisexual.

3D model of the ancestral flower reconstructed by the new study, showing multiple whorls of petal-like organs, in sets of threes. **herve.sauquet@u-psud.fr, juerg.schoenenberger@univ**

Hervé Sauquet, from Université Paris-Sud, France, one of the authors of the paper published this week in Nature Communications said: "There is no living flower that looks exactly like the ancestral one - and why should there be? This is a flower that existed at least 140

million years ago and has had considerable time to evolve into the incredible diversity of flowers that exist today."

We are all familiar with the beauty of flowers - the reproductive structures produced by about 90 % of all living land plants. But their origin and early evolution is a mystery. This is mainly owing to the lack of fossil flowers from the time period when the ancestor of living flowers is thought to have existed.

Dr Jason Hilton from the University of Birmingham, UK, who was not involved in the study, said: "The structure and organisation of the ancestral flower has remained enigmatic.

For instance, we don't know if the oldest flowers were unisexual or bisexual, or if they were pollinated by wind or insects."

Left: Spiral arrangement of petals in a lotus flower. Right: Whorl arrangement of petals in a lily flower. **Science Photo Library**

To reconstruct the appearance of the first flower, the scientists recorded the features - such as the petals and sepals - of the flowers from 792 living species.

They mapped the distribution of these features on to the evolutionary tree of flowering plants enabling them to build a picture of what flowers looked like at key points in their history - including the last common ancestor of all living flowers.

The first flower is reconstructed with petal-like structures arranged in a whorl, so each petal appears in the same plane, like a common lily (but with more whorls), rather than in a spiral, where petals overlap in a spiral arrangement around the stem, like a lotus.

"For some of the features we studied, the result was surprising, especially the fact that organs (such as sepals and petals and the stamens) were probably arranged in whorls instead of spirals, as commonly assumed for the ancestral flower," said Hervé Sauquet.

Sex evolution in flowers has been highly debated. Flowers can be unisexual or bisexual and this study infers a bisexual early flower with both male and female organs.

"This study is important as it tells us how complicated the ancestral flower is likely to be - now the search is on to find it or something closely resembling it in the fossil record. That's if the model is correct - only time (and further study) will tell," said Jason Hilton.

http://wb.md/2uZYkNT

PSA Density: A Better Predictor of Prostate Cancer Risk?

The role of prostate-specific antigen (PSA) density for improving detection of prostate cancer compared with PSA alone

Gerald Chodak, MD August 01, 2017

Hello. I am Dr Gerald Chodak from Medscape. Today I want to talk about the role of prostate-specific antigen (PSA) density for improving detection of prostate cancer compared with PSA alone.

Jue and coworkers[1] reported on nearly 1300 men who were part of a prospective trial evaluating the 4Kscore. They estimated PSA density by performing a standard PSA test, performing an ultrasound, and then using the ellipsoid method for evaluating prostate volume. They divided the patients into three groups based on PSA [level]: < 4 ng/mL, 4-10 ng/mL, and > 10 ng/mL.

For men whose PSA [level] was < 4 ng/mL, density was not a very effective way of improving detection of the disease. However, for men with higher PSA values, they did find a better area under the curve (AUC) when density was compared with the standard PSA method. The problem was, it did not make much of an improvement. For

example, when using PSA density of 0.05 ng/mL/cc, they found that 4% of significant cancers and 8% of the total number of cancers could be missed, while sparing about 15% of men from undergoing a prostate biopsy. The standard PSA density of 0.15 ng/mL/cc missed far too many cancers to be considered worthwhile.

They did find slightly better results if men had had a previously negative biopsy. There, the difference in the AUC between PSA alone and PSA density was the greatest. Whether it is good enough remains to be seen.

Ultimately, what we have here is a tradeoff. Is it worth doing the PSA density to decide whether a biopsy is needed to spare just 15% from having a biopsy while missing some 4%-8% of the cancers? That will be an ongoing discussion between the patient and the doctor.

For now, the bottom line is that in some cases, PSA density might be more useful than PSA alone. I do not think that the tradeoff of improvement in number of men saved from having a biopsy justifies missing 4%-8% of the cancers.

Time will tell. We will have to wait for other studies to further evaluate this. In the past, PSA density was not found to be very useful.[2] Here, the authors are saying [that] if you use a lower density value of 0.05 ng/mL/cc, you can get better results. The question is, are they good enough? I look forward to your comments. Thank you.

Jue JS, Barboza MP, Prakash NS, et al. Re-examining prostate-specific antigen (PSA) density: defining the optimal PSA range and patients for using PSA density to predict prostate cancer using extended template biopsy. Urology. 2017;105:123-128.

Cookson MS, Floyd MK, Ball TP Jr, Miller EK, Sarosdy MF. The lack of predictive value of prostate specific antigen density in the detection of prostate cancer in patients with normal rectal examinations and intermediate prostate specific antigen levels. J Urol. 1995;154:1070-1073.

http://bit.ly/2vsL7xq

UW-Madison study trying to unlock secrets of breast cancer's 'exceptional survivors'

Study of women who continue to live while others die despite the same or better prognosis

Karen Herzog, Milwaukee Journal Sentinel Published 4:15 p.m. CT

DE PERE - Tammy Mocarski remembers her surgeon leaning over her as she woke up in recovery after having what appeared to be a harmless, pea-sized tumor removed from the crease below her left breast. "He wanted to be the one to tell me," she said.

He didn't like the tumor's margins, so he took 15 lymph nodes. Four of the 15 had cancer cells. It was stage 2A breast cancer. And it turned out to be HER2-positive, which tends to be more aggressive and spread more quickly than other cancers.

"My first thought was I'm not going to see my kids grow up," Mocarski recalled, sitting cross-legged on a recliner in her living room and choking back tears as she thought of her four sons, who were between the ages of 2 and 7 at the time. She was only 36.

Sixteen years and six recurrences later — including a metastatic diagnosis in 2006 — Mocarski is surviving long odds. Statistics suggest less than 10% of women diagnosed with metastatic breast cancer will live 10 years or more. But her sons are all in college — the oldest one in medical school — and she is still very much alive.

Is it genetics? Her immune system? Her cancer treatment plan? Lifestyle? Environment?

In the hope of finding an answer, and perhaps helping other women live longer, Mocarski is part of a University of Wisconsin-Madison study of "exceptional survivors" of metastatic breast cancer. These women continue to live while others die despite the same or better prognosis. The study is led by Mark Burkard, an associate professor and breast cancer oncologist at the University of Wisconsin Carbone Cancer Center at UW-Madison.

"If the reason some people are living longer is within our control — treatment, lifestyle or immune response — we could potentially help others be exceptional survivors, too," Burkard said. "There may be things not in our control to modify: specific genetics of these tumors. But then we could predict who would survive longer and we may plan her treatment differently."

Burkard initially enrolled 15 women from Wisconsin in the study, scraping together enough funding from the Carbone Cancer Center to do genetic analysis of their tumors or tissue samples from biopsies.

He has since received $300,000 from the Avon Breast Cancer Crusade and is now scouring the country for 38 more so-called exceptional survivors to study. Ideally, Burkard would add even more to the study if additional research funding could be secured.

DNA analysis is possible because medical facilities are required by law to store tumor and tissue samples for at least 10 years. But genomic analysis of tumor and tissue samples costs $2,000 to $2,600 per sample.

Burkard also will take blood or saliva samples to look at study participants' immune systems, and have them fill out questionnaires about their lifestyle and living environment.

A colleague at the University of Alabama-Birmingham is mining a national database for long-term survivors. An immunology colleague at UW-Madison will look at the immune cells that infiltrate the tumors to examine patients' immune responses to cancer.

"We have a series of anecdotes that some have an exceptional response to treatment," Burkard said. "The more genetically different the tumor is from you, the better the chance the immune system will detect it."

Just last May, the FDA approved the first new drug aimed at the genetic characteristics of a tumor, rather than where it started. Breast cancer might look like a lung cancer or melanoma, for example. So a drug potentially could be repurposed — specifically to the genetics of the tumor — and work as well for breast cancer as it does, say, lung cancer.

"We're starting to say: Rather than define it by the name of where it started, define it by the gene," Burkard said. "We're not completely away from it because some cancers are more likely to have that gene than others."

'I have hunches'

Burkard said studying the most unusual cases, such as exceptional breast cancer survivors, offers the opportunity to learn the most because these cancers are the most extreme in how they behave.

"I have hunches," he said. "My first hunch is there's a set of genes in the cancer that causes them to be slower growing. My fellow scientists heartily disagree and say it's the immune system or environment of the cancer or the treatment. Alcohol, exercise and obesity have been linked with some factors that might be relevant."

Twenty years ago, a mother and daughter who both were in the final months of metastatic breast cancer banked their DNA, as did a younger daughter who assumed she would end up with the disease, too. They hoped a researcher someday could analyze their DNA and learn from it.

"I am glad I'm alive to see that day," said Linda Cook, the daughter from Plover, who didn't have breast cancer at that time but had her blood drawn for DNA testing at the UW-Madison Carbone Cancer Center in anticipation of a diagnosis.

Cook's mother, Myra Hansen of Stevens Point, and sister, Diane Fuller of Traverse City, Mich., both died within a year or so of banking their DNA. They will be part of the study, along with Cook, thanks to their scientific foresight.

Myra Hansen's breast cancer was diagnosed at age 60 — 19 years before she died in 1998. While she was never diagnosed with metastatic cancer, Cook suspects that cancer was in her mother's bones the final year or two because she had back pain. Cook's sister, diagnosed with metastatic breast cancer at age 46, died in 1999 after surviving 12 years — several years longer than most women.

Cook, 64, was diagnosed with breast cancer almost 10 years ago, and metastatic breast cancer about eight years ago. Her chances of surviving beyond five years were roughly 26%. The cancer spread to her spine, ribs and right hip. She currently is in her fifth round of radiation treatment.

"Nothing's going to change for me and my treatment and my lifetime, but how exciting this is if we can help someone else live longer," Cook said of the study. "Perhaps by studying my family's genetic code, we can find that link and metastatic breast cancer would no longer be incurable."

All ages

The study is enrolling women of all ages. Shirley Lake, 85, of Madison was diagnosed with stage 2 breast cancer in 1998. It metastasized 3 ½ years ago. "You're never home free," she said.

Mocarski suspects her treatment and oncologist Michael Volk, have had a lot to do with her survival.

The development of Herceptin — a drug that has improved survival rates for women with stages 1 to 3 HER2-positive breast cancer by more than 30% — had just received FDA approval when Mocarski started taking it. The last two medications she took — each for about four years — had just been approved by the FDA for HER2-positive breast cancer, Mocarski said.

She's currently not taking any cancer medication. But her blood has been tested every four weeks for 16 years because she knows cancer could still in her body.

"There's no indication of metabolic activity right now," she said. "But my first surgeon told me don't ever let them tell you you're in remission. Microscopic cells can be asleep for five, 10, 15 years."

Her longtime oncologist, Volk, plans to retire next year, and she never expected to outlive his career, she said. She met Burkard when the cancer spread to one of her lungs, and saw him again earlier this year, when he asked her to participate in the study.

"I was surprised, flattered and uncomfortable because it was, 'We want to study you because you probably shouldn't be here, but you are,' " said Mocarski.

The walls of her family room feature pictures of her and husband Jay's sons as little boys shortly after her diagnosis, and their high school graduation portraits — pictures she is grateful to see every day, as the boys launch their adult lives in college and beyond.

She still sets short-term goals. She doesn't want to be greedy.

There's no history of breast cancer in Mocarski's family. All four of her boys were born before she turned 35, and she breastfed them. She never smoked.

She tries not to think too much about defying the odds.

"I get a lot of survivor's guilt," she said. "In 16 years, I've met a lot of people, including a couple of friends, who have passed away from the disease, and their prognosis was better than mine. I feel grateful every day."

http://bit.ly/2uadVM1

Increasing productivity by one day each month

UCR, UCLA, and WashU study shows corporate wellness programs lead to increased worker productivity

RIVERSIDE, Calif. -- Corporate wellness programs have been shown to save companies money by reducing absenteeism and health insurance costs.

Researchers at the University of California, Riverside, UCLA, and Washington University in Saint Louis, Mo., have now quantified an additional benefit to companies' bottom line, showing that a wellness program they studied resulted in higher productivity for all participating employees.

This improvement was dramatic: approximately equal to an additional productive work day per month for the average worker.

Titled "Doing Well by Making Well: The Impact of Corporate Wellness Programs on Employee Productivity," the study's first author is Timothy Gubler, an assistant professor of management in the School of Business at UCR. It is forthcoming in the journal Management Science.

Almost 90 percent of companies use some form of corporate wellness programs, with the most comprehensive offering biometric health screenings, nutritional programs, fitness classes, and educational

seminars on topics ranging from smoking cessation to work-life balance.

A recent meta-analysis found that each dollar spent on wellness programs saves $3.27 in health care costs and $2.73 in absenteeism costs.

To quantify the additional benefit to companies through improved motivation and productivity, the current study examined individual productivity and medical data collected over a three-year period at five plants of an industrial laundry company in the Midwestern United States.

The voluntary program was offered to workers, free of charge, at four of the five plants; the fifth did not participate because it used a different health insurance plan, providing a control group for the study.

Employees who signed up for the program (about 85 percent of the staff) were offered access to a simple health exam that included drawing blood, taking blood pressure, and a health survey.

Three weeks later, participants attended an educational seminar where a registered nurse presented them with a personalized health packet detailing their current health status and providing recommendations for improving their health.

About two thirds of employees had a medical condition at the time of screening, according to an analysis by physicians hired by the researchers to evaluate the health data.

After linking the medical data with individual worker productivity data, the authors found that participation in the wellness program increased average worker productivity by over 5 percent--roughly equivalent to adding one additional day of productive work per month for the average employee.

By group, the results showed:

Sick employees whose health improved during the program showed increased productivity levels that averaged 11 percent.

Healthy workers whose health improved during the program showed increased productivity levels that averaged 10 percent.

Healthy employees whose health did not improve during the program showed increased productivity levels that averaged 6 percent.

Sick employees whose health did not improve did not show increased productivity levels.

While unable to precisely identify the mechanisms driving improvements, Gubler said the increase in productivity was consistent with two factors: First, increased employee motivation that stemmed from higher job satisfaction and gratitude from those who discovered an undiagnosed illness; and second, improved capabilities due to improved physical and mental wellness.

Employees who improved exercise and changed their diet saw the biggest increases in productivity.

"By showing concern for workers, organizations can strengthen employees' loyalty and commitment to the company. When workers discover unknown health problems through the program they may feel increased gratitude toward their employer and reciprocate that by increasing their efforts. Additionally, when programs help employees make healthy choices this can positively impact their wellness, mood, energy, and ultimately increase their productivity through increased capability," Gubler said.

Gubler said the findings add to a growing body of management research on the relationship between employee wellbeing and organizational performance.

However, this is the first study to show a direct "causal link" for improvements in productivity through wellness programs, and employees improving their health.

"Our research suggests that corporate wellness plans can boost employee satisfaction by offering a tangible benefit that empowers them to take care of their health in a way that's integrated into their busy lives. The result is healthier and happier employees who are not only less expensive and less absent, but also more productive," he said.

In addition to Gubler, authors on the paper are Ian Larkin of UCLA's Anderson School of Management, and Lamar Pierce of Washington University's John M. Olin School of Business.

http://bit.ly/2htDYaO

In South Asia, humid heat expected to surpass fatal levels by the late 21st century

Deadly heat waves projected in the densely populated agricultural regions of South Asia

Hot and humid temperatures in South Asia, which contains one-fifth of the global population, will exceed the upper limit of human survivability by the late 21st century, scientists project, underscoring an urgent need to adopt alternative strategies on top of those currently proposed to alleviate climate change-induced temperature extremes. In 2015, the fifth deadliest heat wave in recorded history affected large parts of India and Pakistan, claiming around 3,500 lives.

Many studies in South Asia have charted the trajectory of heat waves linked to climate change and their impact on human health; however, the forecast of "wet-bulb temperature," or a measure of temperature, humidity and the human body's ability to cool down in response, is not yet clear.

After running high-resolution simulations under two climate scenarios, Eun-Soon Im and colleagues reveal wet-bulb temperatures are projected to approach the survivability threshold (35 degrees Celsius) over most of South Asia, and exceed it at a few locations, by the end of the century under a business-as-usual (BAU) scenario, while reaching dangerous levels (over 31 Celsius) under a mitigation scenario (roughly comparable to the goals pledged by the 2015 UN Conference on Climate Change).

The authors also found that the population exposed to harmful wet-bulb temperatures will increase from zero in the present day to about 30% under BAU versus only 2% under the mitigation timeline - a substantial difference that points to the significant impact of climate change mitigation efforts. The increase in humid heat raises important questions of environmental justice in agricultural areas where the inhabitants - the majority of whom work outdoors and have poor access to air conditioning - are most vulnerable, the authors say.

http://bit.ly/2vo5mwJ

Acid attack bystanders can make a real difference if they act fast, say experts

Education, legislation and guidance for health professionals, will play a key role in tackling acid attacks

Doctors at Barts Health NHS Trust and the Royal College of Emergency Medicine, say public education, alongside legislation and clear guidance for health professionals, will play a key role in tackling this latest menace on our streets.

In London, the Metropolitan Police recorded almost 300 acid attacks in 2010, after which numbers decreased to 162 in 2012, but demonstrated a steep increase since 2014. There were 454 attacks in 2016, up from 261 in 2015.

Already 2017 has seen a big increase in acid attacks in the UK, relative to 2016. And, whereas in the past most of the attacks were related to robberies, corrosive substances now seem to be a replacement for carrying knives.

Recent figures obtained by the BBC from the Metropolitan Police show that men are twice as likely to be victims of attacks - and many of these attacks have been linked to gang related violent crimes.

Yet the authors point out that currently in the UK, carrying corrosive substances is legal with no restrictions on volume or strength, although a change in legislation is being considered.

In 2002, after similar attacks, Bangladesh banned the open sale of acid and imposed stringent punishment of offenders, which saw the number of attacks fall by 15-20% a year. India and Cambodia have since implemented legislation to combat acid attacks but have yet to introduce laws restricting the ease and availability of acid.

The authors say that bystanders who come to the aid of the victim of an attack "can have an important role in minimising further injury." For example, removing contaminated clothing and washing off the acid with copious amounts of water can minimise scarring and need for surgical reconstruction. Once in the emergency department,

ongoing treatment and specialist review is vital to limit long term physical and emotional effects, they add.

The rising incidence of acid attacks is an evolving challenge to law enforcement, write the authors. Current legislation is being reviewed and may need to be fast tracked to ensure that carrying corrosive substances becomes a criminal offence, they add.

They suggest that "public education is needed on how to deal with these injuries, as immediate treatment can substantially improve the outcome." Similarly, ambulance service responders and health professionals in emergency departments "must have clear guidance on immediate steps to minimise secondary harm and training on how to deal with these devastating, life changing attacks."

http://bbc.in/2v2YB2i

'Exciting discovery' in common cold cure search

Scientists believe they may have made a breakthrough in the search for a cure for the common cold.

Researchers say treatments could be developed based on antimicrobial peptides that occur naturally in the immune systems of humans and animals. The Edinburgh Napier University team observed how they increase the body's natural response to rhinovirus infection. Rhinovirus is the main virus responsible for the common cold.

The team synthesised antimicrobial peptides found in pigs and sheep, and assessed their impact on lung cells infected with rhinovirus. The peptides successfully attacked the virus, and could provide clues for developing novel treatments based on peptides found in nature.

Next steps

Dr Peter Barlow, associate professor of immunology and infection at the university, said: "This is an exciting discovery and our next steps will be to modify the peptide to make it even better at killing this virus. "This research is still in the early stages, but we will ultimately be looking to develop drug treatments that have the potential to cure the common cold." An effective treatment for the cold could help sufferers of more serious lung conditions, such as asthma and chronic obstructive pulmonary disease (COPD), for whom viral infections can pose a serious health risk.

Dr Barlow added: "There is no cure and no vaccine so the development of effective therapies for human rhinovirus, the main causal agent of the common cold and one of the most common causes of viral respiratory tract infections, is an urgent requirement.

"This study represents a major step towards finding a treatment."

Earlier research by Dr Barlow had underlined the potential of antimicrobial peptides in tackling the influenza A virus.

The latest study, was funded by the Chief Scientist Office and medical research charity Tenovus Scotland.

http://bit.ly/2wcZ5Sh

New catalysts efficiently and rapidly remove BPA from water

Approach quickly and cheaply removes over 99% of BPA from water

Carnegie Mellon University chemist Terrence J. Collins has developed an approach that quickly and cheaply removes more than 99 percent of bisphenol A (BPA) from water. BPA, a ubiquitous and dangerous chemical used in the manufacturing of many plastics, is found in water sources around the world.

In a paper published in Green Chemistry, Collins' research team and collaborators at the University of Auckland and Oregon State University also compiled evidence of BPA's presence in a multitude of products and water sources, as well as the chemical's toxicity. The research team builds a strong case for the need to effectively remediate BPA-contaminated water, especially industrial waste streams and landfill runoff, and they offer a simple solution.

BPA is a chemical used primarily in the production of polycarbonate plastic and epoxy resins. Its use is so widespread—BPA can be found in products from DVDs and eyeglass lenses to cash register receipts—to which people and wildlife are regularly exposed.

BPA is dangerous because it mimics estrogen, a naturally occurring hormone, and can affect the body's endocrine system. Studies in fish, mammals and human cells have shown that BPA adversely affects brain and nervous system development, growth and metabolism, and the reproductive system.

Concerns over BPA's health effects prompted manufacturers to start making BPA-free products like baby bottles and water bottles starting in 2010. Ironically, many BPA replacements also have similar toxicity to BPA itself.

"BPA replacements have often not been adequately tested despite the fact that testing is easy to do," said Collins, the Teresa Heinz Professor of Green Chemistry at Carnegie Mellon. "A large team of environmental health scientists and green chemists developed a methodology called the Tiered Protocol for Endocrine Disruption for identifying endocrine disruptors to the highest levels of contemporary science, that we published in Green Chemistry in 2013."

With more than 15 billion pounds of BPA still being produced annually, BPA contamination and cleanup present a significant challenge.

"There is no escape from BPA—for any living creature," said Collins, the Teresa Heinz Professor of Green Chemistry at Carnegie Mellon. "The massive global use of BPA burdens an already overstrained water treatment infrastructure and most BPA water releases simply never reach a water treatment facility. Our approach has high potential to be a much better remediation strategy for BPA-contaminated waste streams."

Currently, BPA-contaminated water such as industrial waste or landfill runoff may or may not be treated before it's released into the environment or to wastewater treatment plants.

Collins' team offers a simple, effective and cheap cleanup solution. Their system involves a group of catalysts called TAML activators, small molecules that mimic oxidizing enzymes. When combined with hydrogen peroxide, TAML activators very effectively break down harmful chemicals in water.

In the current 25-page paper, the researchers demonstrate the efficacy and safety of TAML activators in breaking down BPA. Adding TAMLs and hydrogen peroxide to water heavily contaminated with BPA resulted in a 99 percent reduction of BPA within 30 minutes at near neutral pH, which is the pH norm for wastewater treatment.

TAML treatment at this pH caused BPA to assemble into larger units called oligomers, which clump together and precipitate out of the water. According to Collins, the oligomers could be filtered and disposed of in a BPA water treatment facility. Most importantly, extensive studies by Collins and his collaborators found that the oligomers are themselves not harmful. The nature of the bonds that stick the BPA molecules together doesn't allow the oligomers to revert to BPA.

To ensure the safety of the decontaminated water, including the oligomers, the researchers tested it with Tiered Protocol for Endocrine Disruption (TiPED) assays. They found that the TAML-treated BPA water did not show estrogen activity or cause abnormalities in yeast and developing zebrafish embryos.

The researchers also tested the efficacy of TAML treatment on BPA-laden water at a pH of 11. At this higher pH, there was a greater than 99.9 percent reduction in BPA within 15 minutes. In contrast with pH 8.5 treatment, the BPA molecules were destroyed and no oligomers were detected.

"Because TAML/hydrogen peroxide treatment eliminates BPA from water so easily at concentrations that are similar to a variety of waste streams including paper plant processing solutions and landfill leachate, assuming the lab studies transfer to the real world, we can now offer a new and simple procedure for reducing BPA exposures worldwide," Collins said.

More information: Yusuf Onundi et al, A Multidisciplinary Investigation of the Technical and Environmental Performances of TAML/Peroxide Elimination of Bisphenol A Compounds

from Water: Destruction, Oligomerisation, Mechanism, End Product Toxicity, and Applications, Green Chem. (2017). DOI: 10.1039/c7gc01415e

http://bit.ly/2v2T0JH

Statins reduce chance of second stroke by 30%: study

Statins should be a lifelong therapy for ischemic stroke patients, say Taiwanese researchers

For people who have already had a stroke, knocking off the statins is a really bad idea, research from Taiwan has confirmed.

In a paper published in the Journal of the American Heart Association, scientists led by Meng Lee of Chang Gung University College of Medicine looked at the health of 45,151 Taiwanese patients who experienced an ischemic stroke – caused by a build-up of cholesterol in the arteries, restricting blood flow to the brain – between 2001 and 2012. All were prescribed statins – which work by reducing the clogging of arteries – within 90 days of being discharged from hospital. The study has found that patients who stopped taking the medication three to six months after discharge ran a much higher risk of having a second stroke, as well as a substantially higher risk of dying from other causes.

Lee and his team used data from the Taiwan National Health Insurance program, which covers 99% of the country's population.

Patients who stopped taking the medication had a 42% increased risk of having a second stroke, and a 37% risk of dying from other causes, the researchers found. Interestingly, patients who shifted from a high to a lower dose of statins did not have an increased risk of a second event. The results strongly indicate statins should not be regarded as a short-term or interim medication, Lee says.

"Based on our findings of this large group of patients in the 'real world', we believe that statins should be a lifelong therapy for ischemic stroke patients if a statin is needed to lower the patient's cholesterol," he says. "Discontinuation of statin treatment in patients with ischemic stroke should be strongly discouraged in any stage -- acute or chronic -- of stroke."

http://nyti.ms/2vw18Tx

Lyme Disease's Worst Enemy? It Might Be Foxes

The rise in tick-borne disease may be tied to a dearth of traditional mouse predators

By AMY HARMON AUG. 2, 2017

It is August, the month when a new generation of black-legged ticks that transmit Lyme and other diseases are hatching. On forest floors, suburban estates and urban parks, they are looking for their first blood meal. And very often, in the large swaths of North America and Europe where tick-borne disease is on the rise, they are feeding on the ubiquitous white-footed mice and other small mammals notorious for harboring pathogens that sicken humans.

A red fox, left, and a stone marten, right, are both traditional predators of mice and other creatures that carry the harmful germs that ticks pass on to spread illnesses like Lyme disease. **Credit Left: Eva and Helmut Pum/McPhoto, via Ullstein Bild, via Getty Images; right: Reiner Bernhardt, via picture-alliance, via DPA, via Associated Press**

But it doesn't have to be that way. A new study suggests that the rise in tick-borne disease may be tied to a dearth of traditional mouse predators, whose presence might otherwise send mice scurrying into their burrows. If mice were scarcer, larval ticks, which are always born uninfected, might feed on other mammals and bird species that do not carry germs harmful to humans. Or they could simply fail to find that first meal. Ticks need three meals to reproduce; humans are at risk of contracting diseases only from ticks that have previously fed on infected hosts.

For the study, Tim R. Hofmeester, then a graduate student at Wageningen University in the Netherlands and the lead researcher of the study, placed cameras in 20 plots across the Dutch countryside to

measure the activity of foxes and stone martens, key predators of mice. Some were in protected areas, others were in places where foxes are heavily hunted.

Over two years, he also trapped hundreds of mice — and voles, another small mammal — in the same plots, counted how many ticks were on them, and tested the ticks for infection with Lyme and two other disease-causing bacteria. To capture additional ticks, he dragged a blanket across the ground.

In the plots where predator activity was higher, he found only 10 to 20 percent as many newly hatched ticks on the mice. Thus, there would be fewer ticks to pass along pathogens to next generation of mice. In the study, the density of infected "nymphs," as the adolescent ticks are called, was at 15 percent of levels in areas where foxes and stone martens were less active.

"The predators appear to break the cycle of infection,'' said Dr. Hofmeester, who earned his Ph.D. after the study.

Despite stuffing his pant legs into his socks and using permethrin, a tick repellent, he said he removed more than 100 ticks from his own body.

Interestingly, the predator activity in Dr. Hofmeester's plots did not decrease the density of the mouse population itself, as some ecologists had theorized it might. Instead, the lower rates of infected ticks, Dr. Hofmeester suggested in the paper, published in Proceedings of the Royal Society B, may be the result of small mammals curtailing their own movement when predators are around.

"This is the first paper to empirically show that predators are good for your health with respect to tick-borne pathogens," said Dr. Taal Levi, an ecologist at Oregon State University who was not involved in the study. "We've had the theory but this kind of field work is really hard and takes years." He also said of Dr. Hofmeester, "Wow, I have to send him an email."

Habitat fragmentation, hunting and the removal of larger predators like cougars may all figure into the dwindling of small mammal predators like foxes, weasels, fishers and martens, Dr. Levi said. If the study's results are borne out by more research, public health officials might be moved to try interventions like protecting foxes or factoring the habitat needs of particular predators into land-use decisions to foster their population size. Nothing else — like culling deer or spraying lawns with tick-killing pesticide — has worked so far to stem the incidence of tick-borne disease, which is spreading in the Midwestern United States, in parts of Canada and at higher altitudes across Europe.

"The takeaway is, we shouldn't underestimate the role predators can play in reducing Lyme disease risk," said Richard S. Ostfeld, a senior scientist at the Cary Institute of Ecosystem Studies, who originally speculated on the importance of small mammal predators in a 2004 paper. "Let's not discount these cryptic interactions that we don't see very often unless we put camera traps in the woods."

Correction: August 3, 2017

Because of an editing error, an earlier version of this article incorrectly described the number of newly hatched ticks found on mice in areas of a study where predator activity was higher. It was 10 to 20 percent, not 5 to 10 percent. The density of infected adolescent ticks in areas where foxes and stone martens were active was also incorrectly described. It was 15 percent, not 6 percent, of levels in areas where foxes and stone martens were less active.

http://bit.ly/2wrDAfA

Scientists deliver knockout blow to multiple cancers

Targeting healthy cells that have been hijacked by cancer cells could help treat many different types of the disease, according to research* funded by Cancer Research UK and published in the Journal of the National Cancer Institute today (Thursday).

Scientists found that targeting an enzyme known as NOX4 stops the action of a type of cell called cancer associated fibroblasts (CAFs), reducing the size of tumours in mice by up to 50 per cent.**

Fibroblasts are healthy cells whose role is to hold different types of organs together. When they are hijacked by cancer cells, they become CAFs and are known to help tumours grow, spread and evade therapy. Until now, attempts to target them have proved unsuccessful.

In line with previous studies, the team at the University of Southampton found that higher levels of CAFs were associated with poorer survival in several cancers including bowel, head and neck cancers.[***]

For the first time, they identified that NOX4[****] is needed for CAFs to form and help tumours grow in many cancer types. But they could stop this happening by blocking NOX4 using a drug that is being developed to treat a condition called organ fibrosis.

These findings could form the basis for new treatments and help make cancers respond better to existing drugs. Cancer Research UK is now funding the Southampton scientists to see if this approach improves treatments like immunotherapy and chemotherapy to make them more effective.

Professor Gareth Thomas, lead researcher and Chair of Experimental Pathology at the University of Southampton, said: "By looking at many types of cancer, we have identified a common mechanism responsible for CAF formation in tumours.

"These cells make cancers aggressive and difficult to treat, and we can see exciting possibilities for targeting CAFs in many patients who don't respond well to existing therapies."

Dr Áine McCarthy, Cancer Research UK's senior science information officer, said: "Some cancers are incredibly difficult to treat, and can use the body's own cells to help them grow, evade treatment and spread around the body. Researchers have been trying to unlock the secrets behind this for many years and this study is a big step forward in understanding how some cancers achieve this.

"These findings show that CAFs can be targeted with a drug and their 'pro-tumour' effects can be reversed in mice, giving researchers a starting point to develop new and potentially more effective treatments in the future."

Notes to editor:

[*]*Hanley, C, J., et al., Targeting the myofibroblastic cancer-associated fibroblast phenotype through inhibition of NOX4. Journal of the National Cancer Institute.*

[**]*mice were treated pharmacologically with a drug called GKT137831 to inhibit NOX4, which statistically significantly reduced myofibroblast accumulation (68.4%, 95%CI=14.6-122.3%, p=0.02) and tumour growth (46.8%, 95%CI=15.9-77.8%, p=0.006).*

[***]*CAF accumulation and prognostic significance in head & neck cancer (oral, n=260; oropharyngeal, n=271), and colorectal cancer (n=56) was analysed using immunohistochemistry. Patients with moderate/high levels of myofibroblastic-CAF had a statistically significant decrease in survival rates in each cancer type analysed.*

[****]*CAF formation was dependent on the generation of intracellular reactive oxygen species, by NOX4 (NAD (P)H Oxidase). A statistically significant increase in NOX4 expression was found in multiple human cancers. CAFs remain poorly understood, and clinically effective treatments targeting CAFs are yet to be developed.*

http://bit.ly/2wrGQb4

Humans have been altering tropical forests for at least 45,000 years

Tens of thousands of years of controlled burns, forest management and clear-cutting have implications for modern conservation efforts and shatter the image of the 'untouched' tropical forest

The first review of the global impact of humans on tropical forests in the ancient past shows that humans have been altering these environments for at least 45,000 years. This counters the view that tropical forests were pristine natural environments prior to modern agriculture and industrialization. The study, published today in Nature Plants, found that humans have in fact been having a dramatic impact on such forest ecologies for tens of thousands of years, through techniques ranging from controlled burning of sections of forest to plant and animal management to clear-cutting. Although previous studies had looked at human impacts on specific tropical forest locations and ecosystems, this is the first to synthesize data from all over the world.

The paper, by scientists from the Max Planck Institute for the Science of Human History, Liverpool John Moores University, University College London, and École française d'Extrême-Orient, covered three distinct phases of human impact on tropical forests, roughly correlating to hunting and gathering activities, small-scale agricultural activities, and large-scale urban settlements.

Big impacts of small hunter-gatherer groups

In the deep past, groups of hunter-gatherers appear to have burned areas of tropical forests, in particular in Southeast Asia as early as 45,000 years ago, when modern humans first arrived there. There is evidence of similar forest burning activities in Australia and New Guinea. By clearing parts of the forest, humans were able to create more of the "forest-edge" environments that encouraged the presence of animals and plants that they liked to eat.

There is also evidence, though still debated, that these human activities contributed to the extinction of forest megafauna in the Late Pleistocene (approximately 125,000 to 12,000 years ago), such as the giant ground sloth, forest mastodons, and now-extinct large marsupials. These extinctions had significant impacts on forest density, plant species distributions, plant reproductive mechanisms, and life-cycles of forest stand, that have persisted to the present day.

Farming the forest

The earliest evidence for farming in tropical forests is found in New Guinea, where humans were tending yam, banana and taro by the Early-Mid Holocene (10,000 years ago). Early farming efforts in tropical forests, supplemented by hunting and gathering, had significant consequences. Humans domesticated forest plants and animals, including sweet potato, chili pepper, black pepper, mango, banana and chickens, altering the forest ecologies and contributing significantly to global cuisine today.

In general, when groups employed indigenous tropical forest agricultural strategies based on local plants and animals, these did not result in significant or lasting damage to the environment. "Indeed, most communities entering these habitats were initially at low population densities and appear to have developed subsistence systems that were tuned to their particular environments," states Dr. Chris Hunt of Liverpool John Moores University, a co-author of the study.

However, as agricultural intensity increased, particularly when external farming practices were introduced into tropical forests and island environments, the effects became less benign. When agriculturalists bringing pearl millet and cattle moved to the area of tropical forests in western and central Africa about 2,400 years ago, significant soil erosion and forest burning occurred. Similarly, in Southeast Asia, large areas of the tropical forests were burned and cleared from c. 4,000 years ago following the arrival of rice and millet farming. For example, the increase in demand for palm oil has led to clear-cutting of tropical forests to make room for palm plantations. "These practices, which induce rampant clearance, reduce biodiversity, provoke soil erosion, and render landscapes more susceptible to the outbreak of wild fires, represent some of the greatest dangers facing tropical forests," notes Hunt.

Sprawling cities in the jungle

Despite previous notions of tropical forests as "green deserts" not suitable for human habitation, recent discoveries using new technologies have shown that ancient populations created vast urban settlements in these habitats. New data, including surveys made with canopy-penetrating Light Detection and Ranging (LiDAR) mapping, have revealed human settlement in the Americas and Southeast Asia on a scale that was previously unimagined. "Indeed, extensive settlement networks in the tropical forests of Amazonia, Southeast Asia, and Mesoamerica clearly persisted many times longer than more recent industrial and urban settlements of the modern world have so far been present in these environments," notes Dr. Patrick Roberts of the Max Planck Institute for the Science of Human History, lead author of the paper.

Lessons can be learned from how these ancient urban centers dealt with environmental challenges that are still faced by modern cities in these areas today. Soil erosion and the failure of agricultural systems necessary to feed a large population are problems encountered by large urban centers, past and present. In some Mayan areas, urban populations "gardened" the forest, by planting a variety of complementary food crops in and around the existing forest rather

than clearing it. On the other hand, other groups appear to have over-stressed their local environments through forest clearing and monoculture planting of corn, which, in combination with climate change, resulted in dramatic population declines.

Another interesting finding is that ancient forest cities showed the same tendency towards sprawl as is now being recommended by the architects of modern cities in these zones. In some cases these extensive urban fringes appear to have provided a sort of buffer-zone, helping to protect the urban centers from the effects of climate change and providing food security and accessibility. "Diversification, decentralization and 'agrarian urbanism' appear to have contributed to overall resilience," states Dr. Damian Evans, a co-author of the paper. These ancient forest suburbs are now being studied as potential models of sustainability for modern cities.

Lessons for the future

The global data compiled for this paper shows that a pristine, untouched tropical forest ecosystem does not exist - and has not existed for tens of thousands of years. There is no ideal forest environment that modern conservationists can look to when setting goals and developing a strategy for forest conservation efforts. Rather, an understanding of the archaeological history of tropical forests and their past manipulation by humans is crucial in informing modern conservation efforts. The researchers recommend an approach that values the knowledge and cooperation of the native populations that live in tropical forests. "Indigenous and traditional peoples - whose ancestors' systems of production and knowledge are slowly being decoded by archaeologists - should be seen as part of the solution and not one of the problems of sustainable tropical forest development," states Roberts. The researchers also emphasize the importance of disseminating the information learned from archaeology to other disciplines. By working together, these groups can help to establish a better understanding of the tropical forest environments and how best to protect them.

http://bit.ly/2fjCKOX

Nano aluminium offers fuel cells on demand – just add water

Hydrogen could provide an alternative to battery power

By David Hambling

The accidental discovery of a novel aluminium alloy that reacts with water in a highly unusual way may be the first step to reviving the struggling hydrogen economy.

It could offer a convenient and portable source of hydrogen for fuel cells and other applications, potentially transforming the energy market and providing an alternative to batteries and liquid fuels.

“The important aspect of the approach is that it lets you make very compact systems,” says Anthony Kucernak, who studies fuel cells at Imperial College London and wasn’t involved with the research.

“That would be very useful for systems which need to be very light or operate for long periods on hydrogen, where the use of hydrogen stored in a cylinder is prohibitive.”

The discovery came in January, when researchers at the US Army Research Laboratory at Aberdeen Proving Ground, Maryland, were working on a new, high-strength alloy, says physicist Anit Giri. When they poured water on it during routine testing, it started bubbling as it gave off hydrogen.

That doesn’t normally happen to aluminium. Usually, when exposed to water, it quickly oxidises, forming a protective barrier that puts a stop to any further reaction. But this alloy just kept reacting. The team had stumbled across the solution to a decades-old problem.

Hydrogen has long been touted as a clean, green fuel, but it is difficult to store and move around because of its bulk. “The problem with hydrogen is always transportation and pressurisation,” says Giri.

Slow reaction

If aluminium could be made to effectively react with water, it would mean hydrogen on demand. Unlike hydrogen, aluminium and water are easy to carry – and both are stable.

But previous attempts to drive the reaction required high temperatures or catalysts, and were slow: obtaining the hydrogen took hours and was around 50 per cent efficient.

The new alloy, which the team is in the process of patenting, is made of a dense powder of micron-scale grains of aluminum and one or more other metals arranged in a particular nanostructure. Adding water to the mix produces aluminium oxide or hydroxide and hydrogen – lots of it.

“Ours does it to nearly 100 per cent efficiency in less than 3 minutes,” says team leader Scott Grendahl. Moreover, the new material offers at least an order of magnitude more energy than lithium batteries of the same weight. And unlike batteries, it can remain stable and ready for use indefinitely.

The army team has used the material to power a small, radio-controlled tank. Grendahl doesn’t see any practical issues with scaling up production to produce hundreds of tonnes of the stuff as it can be made from scrap aluminium, which is relatively cheap. The new material could power everything from laptops to buses and cars.

“In principle, the process should work,” says Robert Steinberger-Wilckens, who directs a fuel cell programme at the University of Birmingham, UK.

But he cautions that a repeat experiment is needed to show that the reaction works the way it should. “There’s a lot of stuff that works in the laboratory but not in the field.”

If it does pan out, the powder could also be used as the raw material for 3D printing. The researchers have put forward proposals – now being considered by the army – for small air or ground robots that use their own structure as fuel.

These self-cannibalising machines would be useful for one-way intelligence-gathering missions, burning themselves up at the end to leave no trace.

http://bit.ly/2fjMdFV

Breakthrough by scientists finds arthritis drug could treat blood cancer patients

Blood cancer sufferers could be treated with a simple arthritis drug, scientists at the University of Sheffield have discovered

Blood cancer sufferers could be treated with a simple arthritis drug, scientists at the University of Sheffield have discovered.

Every year 3,000 people in the UK are diagnosed with Polycythemia Vera (PV), a type of blood cancer which causes an overproduction of red blood cells. Patients suffer with itching, headaches, weight loss, fatigue and night sweats. Current treatments do not slow the disease progression and provide little relief from symptoms.

Dr Martin Zeidler, from the University of Sheffield's Department of Biomedical Science, working with colleagues from the Department of Haematology at the Royal Hallamshire Hospital, and funded by the Medical Research Council (MRC) have discovered that methotrexate (MTX) - a drug on the World Health Organisation list of essential medicines and commonly used to treat arthritis - works by directly inhibiting the molecular pathway responsible for causing disease.

Initial tests were carried out on fruit fly cells to screen for small molecules that modulate JAK/STAT signalling - a signalling pathway whose misregulation is central to the development in humans of Myeloproliferative neoplasms (MPNs), the collective term for progressive blood cancers like PV.

Further testing in human cells showed that methotrexate acts as a potent suppressor of JAK/STAT pathway activation - even in cells carrying the mutated gene responsible for MPNs in patients.

Dr Martin Zeidler said the latest tests on mice were entirely consistent with the cell-based studies.

The tests showed low-dose MTX suppresses JAK/STAT pathway activity and is able to normalise both the raised blood counts and the increase in spleen size associated with the disease in these mice.

"We have now shown pretty conclusively that we can use this approach to treat mouse models of human MPNs, results which provide a much more tangible prospect of success in humans," he said. "Repurposing MTX has the potential to provide a new, molecularly targeted treatment for MPN patients within a budget accessible to healthcare systems throughout the world - a development that may ultimately provide substantial clinical and health economic benefits."

MTX has been used for 35 years to treat inflammatory diseases including rheumatoid arthritis, Crohn's disease and psoriasis. Even though the mechanisms by which MTX acts in these diseases had not previously been understood, the safety and effectiveness of MTX is well documented and many millions of patients regularly take the drug. Strikingly, diseases such as rheumatoid arthritis all feature inflammatory processes driven by JAK/STAT activity and the effectiveness of MTX in these inflammatory diseases may well be a consequence of its ability to dampen the JAK/STAT pathway.

The team now hope to go on to a full clinical trial early next year.

The research paper Low-dose methotrexate in myeloproliferative neoplasm models was published in Haematologica, the journal of the European Hematology Association and the Ferrata Storti Foundation.

http://bit.ly/2hzN06p

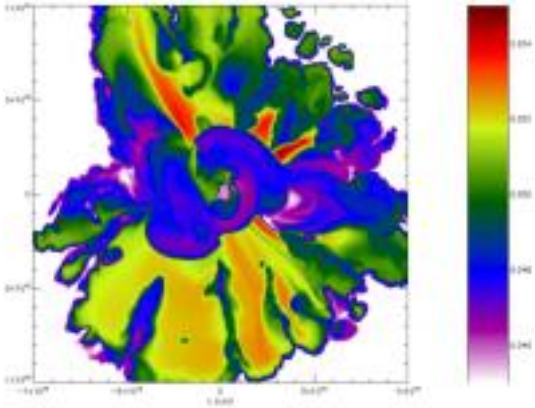
Our solar system's 'shocking' origin story

New work offers fresh evidence supporting the supernova shock wave theory of our solar system's origin

Washington, DC-- According to one longstanding theory, our Solar System's formation was triggered by a shock wave from an exploding supernova. The shock wave injected material from the exploding star into a neighboring cloud of dust and gas, causing it to collapse in on itself and form the Sun and its surrounding planets.

New work from Carnegie's Alan Boss offers fresh evidence supporting this theory, modeling the Solar System's formation beyond the initial cloud collapse and into the intermediate stages of star formation. It is published by the Astrophysical Journal.

One very important constraint for testing theories of Solar System formation is meteorite chemistry. Meteorites retain a record of the elements, isotopes, and compounds that existed in the system's earliest days. One type, called carbonaceous chondrites, includes some of the most-primitive known samples.



The colors represent the relative amounts of short-lived radioactive isotopes, such as iron-60, injected into a newly formed protoplanetary disk (seen face on with the protostar being the light purple blob in the middle) by a supernova shock wave. **Alan Boss**

An interesting component of chondrites' makeup is something called short-lived radioactive isotopes. Isotopes are versions of elements with the same number of protons, but a different number of neutrons. Sometimes, as is the case with radioactive isotopes, the number of neutrons present in the nucleus can make the isotope unstable. To gain stability, the isotope releases energetic particles, which alters its number of protons and neutrons, transmuting it into another element.

Some isotopes that existed when the Solar System formed are radioactive and have decay rates that caused them to become extinct within tens to hundreds of millions of years.

The fact that these isotopes still existed when chondrites formed is shown by the abundances of their stable decay products--also called daughter isotopes--found in some primitive chondrites.

Measuring the amount of these daughter isotopes can tell scientists when, and possibly how, the chondrites formed.

A recent analysis of chondrites by Carnegie's Myriam Telus was concerned with iron-60, a short-lived radioactive isotope that decays into nickel-60.

It is only created in significant amounts by nuclear reactions inside certain kinds of stars, including supernovae or what are called asymptotic giant branch (AGB) stars.

Because all the iron-60 from the Solar System's formation has long since decayed, Telus' research, published in Geochimica et Cosmochimica Acta, focused on its daughter product, nickel-60.

The amount of nickel-60 found in meteorite samples--particularly in comparison to the amount of stable, "ordinary" iron-56--can indicate how much iron-60 was present when the larger parent body from which the meteorite broke off was formed.

There are not many options for how an excess of iron-60--which later decayed into nickel-60--could have gotten into a primitive Solar System object in the first place--one of them being a supernova.

While her research did not find a "smoking gun," definitively proving that the radioactive isotopes were injected by a shock wave, Telus did show that the amount of Fe-60 present in the early Solar System is consistent with a supernova origin.

Taking this latest meteorite research into account, Boss revisited his earlier models of shock wave-triggered cloud collapse, extending his computational models beyond the initial collapse and into the intermediate stages of star formation, when the Sun was first being created, an important next step in tying together Solar System origin modeling and meteorite sample analysis.

"My findings indicate that a supernova shock wave is still the most-plausible origin story for explaining the short lived radioactive isotopes in our Solar System," Boss said.

Boss dedicated his paper to the late Sandra Keiser, a long-term collaborator, who provided computational and programming support at Carnegie's Department of Terrestrial Magnetism for more than two decades. Keiser died in March.

The software used in this research was in large part developed by the DOE-supported ASC/Alliances Center for Astrophysical Thermonuclear Flashes at the University of Chicago.

http://bit.ly/2vCzi86

CRISPR skin grafts could replace insulin injections for diabetes

Genetically modified skin grafts have protected mice from developing diabetes

By Rachel Baxter

Genetically modified skin grafts have protected mice from developing diabetes, suggesting the technique may help people with the condition.

The method makes use of the gene that encodes a hormone called glucagon-like peptide-1 (GLP-1). This hormone decreases appetite and helps regulate blood sugar levels by triggering the release of insulin, which removes excess glucose from the blood.

However, the hormone only works for a short period. To tackle this, Xiaoyang Wu at the University of Chicago, Illinois, and his colleagues used CRISPR gene-editing to alter the GLP-1 gene so it would make a hormone that is active in the blood for longer. They then inserted this gene into mouse skin cells in a dish, and developed them into skin grafts that could be transplanted onto mice, letting the modified hormone get into their blood.

Skin therapy

The grafts were given to mice that were fed a high-fat diet. These mice went on to gain around half as much weight as those not given grafts, and developed less resistance to insulin. High insulin resistance is a common precursor to type 2 diabetes.

The researchers gained similar results when they made the grafts out of human skin and transplanted them onto hairless mice.

The edited gene was found to make GLP-1 hormones that were stable and active for three months, suggesting that grafts could be a desirable alternative to daily insulin injections.

It will be relatively easy to translate this into a treatment for people, because skin grafts have been used to treat burn wounds for many years, says Wu. He suggests that grafts with different edited genes may be useful for treating other types of disease too.

"I do predict that gene and cell therapies will ultimately replace repeated injections for the treatment of chronic diseases," says Timothy Kieffer at the University of British Columbia in Vancouver, Canada.

Journal reference: Cell Stem Cell, DOI: 10.1016/j.stem.2017.06.016

http://bit.ly/2uwAupl

We may finally be able to slow Parkinson's, with a diabetes drug

Diabetes drug can slow the progress of Parkinson's disease, and seems to target the underlying cause of the condition

By Jessica Hamzelou

A diabetes drug can slow the progress of Parkinson's disease, and seems to target the underlying cause of the condition, not just its symptoms. The finding adds weight to the theory that the two conditions work in a similar way.

Parkinson's disease leads to the loss of brain cells that make dopamine, a chemical that helps control body movements. Standard treatments for the condition try to replace the missing dopamine. "That goes a good way to improving symptom control, but it does nothing for the underlying disease pathology," says Tom Foltynie of the National Hospital for Neurology and Neurosurgery in London.

There is still no cure for Parkinson's. While replacing dopamine can improve the tremors and stiffness, it doesn't stop the brain from continuing to deteriorate. In an attempt to slow this, Foltynie and his colleagues have turned to a drug typically used to treat type 2 diabetes, called exenatide.

This drug comes from a class of compounds originally isolated from the venom of a lizard called the gila monster. Not only can these help control blood sugar levels – which is useful for people with diabetes – this kind of drug also seems to protect neurons from toxins.

Improved symptoms

Foltynie's colleagues gave exenatide to 31 people with moderate Parkinson's disease over 48 weeks. The participants injected themselves with the drug every day – except the day before assessments – while 29 people with a similar level of disease did the same with a placebo.

Eight weeks after the end of the trial, Foltynie's team assessed all the volunteers' symptoms. Those who had received the placebo had deteriorated over the course of the year – by an average of three points on a 200-point scale, a typical rate of decline for people with Parkinson's.

But those who had taken exenatide showed an average improvement of one point, putting them, on average, four points ahead of the placebo group.

Brain scans also showed that those taking the drug showed less degeneration. However, these effects were subtle – they didn't perform better on cognitive tests, and there wasn't a visible improvement in their day-to-day symptoms.

Shared roots

"You can say with a reasonable level of certainty that the drug is slowing the disease down, if only by a small amount," says David Dexter, a neuropharmacologist at the charity Parkinson's UK.

The fact that a diabetes drug seems to help Parkinson's adds to a growing body of research suggesting that neurodegenerative diseases like Alzheimer's and Parkinson's may work in a similar way to diabetes, and that neurons can become unresponsive to insulin in the same way that cells in the pancreas do in type 2 diabetes.

This can affect how cells produce energy, causing them to starve and leading to inflammation. Exenatide and drugs like it seem to protect cells from this damage, says Foltynie.

Exenatide is thought to be generally safe, although its side effects can include an upset stomach and weight loss.

The drug isn't yet recommended for Parkinson's, says Foltynie. "We need to remain scientific and have proof that the benefits outweigh the risks."

Journal reference: The Lancet, DOI: 10.1016/S0140-6736(17)31585-4

http://bit.ly/2uhd9wK

Math professor thinks drugs are too damn high — so he's teaching people how to make pharmaceuticals at home

A math professor was part of a human rights envoy to Central America when he went to a pharmacy and discovered there was no birth control. The concept was so absurd to him that he decided people should make it themselves.

Sarah K. Burris

Dr. Mixael Laufer **(Photo: Twitter)**

"I thought to myself, 'This is a country where there are there are methamphetamine and ecstasy labs everywhere. Birth control isn't that much more complicated,'" Dr. Mixael Laufer told Gizmodo in an interview. "'Why aren't these people just making their own birth control?'"

While Laufer works full time teaching math, his hobby is chemistry. Like something out of the show "Breaking Bad," he decided he could use his chops to help people. In 2015, he came together with hackers and scientists to found the Four Thieves Vinegar, dedicated to starting a DIY revolution for legal drugs.

"People should be able to take control of their own health choices," he said. "If you work within an external regulatory structure, you have no choice. It is a black box you cannot audit."

He brought up the epinephrine (EpiPen) auto-injectors by Mylan Pharmaceuticals, which significantly increased the price arbitrarily. The backlash was swift, the price eventually came down and congressional hearings were called. In the meantime, the internet exploded with ways for people to build their own homemade EpiPen. There are many other examples.

When "pharma bro" Martin Shkreli jacked up the price of an HIV/AIDS medication from $13 to $750, Laufer got to work. He recently uploaded instructions on how to make Daraprin at home for significantly less. In another case it was the insurer that was was preventing life-saving cures. United HealthCare was called out Wednesday for denying coverage of HIV/AIDS medication to a man because he is gay.

This is part of the reason Laufer thinks capitalism is the larger villain to access to medicine. He ultimately wants to design a kind of "easy bake oven" for making standard pharmaceuticals at home.

"This stuff should be no more difficult than Ikea furniture to put together," he said of the project that is still in its early stages. "There is a list of parts to order, and then you just have to 3D-print this one part. You can just upload the design and order that part online."

He hopes to develop a list of things to purchase to build a kind of "home chemistry kit" for everyday people to use for legal drug-making. The FDA is having a mini-meltdown at the idea.

"Using unapproved prescription drugs for personal use is a potentially dangerous practice," an FDA spokesperson told Gizmodo. "Neither the FDA nor the American public have any assurance that unapproved products are effective, safe or produced under current good manufacturing practices. Unapproved drugs may be contaminated, sub-potent, super-potent or counterfeit."

The FDA has drawn fire recently for their approval of drugs that had safety problems. In fact, one in three drugs approved by the FDA were considered hazardous. In 2015, they were criticized for essentially approving everything, regardless of the dangers of the drugs. Researchers also found recently that medication that had already reached the FDA's expiration date could still work despite being considered "old" by the agency.

Jeremiah Johnson, who takes part in an MIT research group, cautioned that it would be easy to end up making the wrong drug or dosing it incorrectly. "It would not be a good idea to DIY pharmaceuticals," he told Gizmodo. "There is no way that I can see to make this process recommended," Johnson told Gizmodo.

Still, Laufer said that he's made his own aspirin and survived after taking it. "People who think this is unsafe should think about it for more than two seconds," he said. "If someone is dying of a chronic disease, there's already not much worse they can get."

He admits that things can go wrong if someone does it incorrectly but things can also go wrong if the drugs are from the pharmacy.

"But when someone doesn't have access to an essential medicine, what's left in terms of their options?" he asked.

http://bit.ly/2ufzIh6

Fido And Fluffy Are Ruining The Environment, UCLA Study Says

America's beloved dogs and cats play a significant role in causing global warming, according to a new study by UCLA.

By Paige Austin (Patch Staff)

kitty.green66 via flickr.com

LOS ANGELES, CA — When it comes to global warming, Fido and Fluffy are part of the problem, a new study by UCLA indicates.

Most cat or dog lovers would say they can't imagine living in a world without pets, but as the threat of global warming increases, environmentally conscious pet lovers may need to make some tough choices, according to the study.

Pet ownership in the United States creates about 64 million tons of carbon dioxide a year, UCLA researchers found. That's the equivalent of driving 13.6 million cars for a year. The problem lies with the meat-filled diets of kitties and pooches, according to the study by UCLA geography professor Gregory Okin.

Dogs and cats are responsible for 25 to 30 percent of the impacts of meat production in the United States, said Orkin. Compared to a plant-based diet, meat production "requires more energy, land and water and has greater environmental consequences in terms of erosion, pesticides and waste," the study found.

And what goes in, must come out. In terms of waste, Okin noted, feeding pets also leads to about 5.1 million tons of feces every year, roughly equivalent to the total trash production of Massachusetts.

"Given the significant environmental impact of meat production, the contributions of our omnivorous and carnivorous pets deserve special attention," according to Okin's study, published in the journal PLOS ONE. "The U.S. has the largest population of pet dogs and cats globally, with an estimated 77.8 million dogs and 85.6 million cats in 2015."

While previous studies have examined the impact of pet ownership on carbon use, water quality, disease and wildlife, Okin's study delved into its impact on total U.S. energy and meat consumption and the environmental impact of that consumption.

"This analysis does not mean to imply that dog and cat ownership should be curtailed for environmental reasons, but neither should we view it as an unalloyed good," Okin wrote in the study. "It is clear that a transition to pets that eat less meat, and therefore have less environmental impact, would reduce the overall U.S. consumption of meat."

Okin's report notes the social and emotional benefits of owning dogs and cats, insisting the study is intended to increase the awareness of the impact such pets have on the nation's meat-production industry and its environmental effects.

"Additional research is needed to evaluate the animal content and human-edibility of ingredients in dog and cat food after processing, but the calculations presented here indicate that these pets comprise a significant proportion of U.S. energy and animal-derived product consumption, with the consequent environmental impacts, including greenhouse gas emission and feces production," he wrote.

Okin noted that the pet food industry has made advancements in manufacturing, product design and alternative protein sources, but more can be done.

Simple measures like feeding domestic dogs and cats nutritionally appropriate amounts will certainly reduce their environmental and energetic impact, Okin wrote. "However, without large-scale reduction in their number and changes to the food system that drastically reduces the per-capita animal product consumption, the environmental and energetic impact of these animals will remain significant."

http://bit.ly/2uhhDmY

Recreating the wild: De-extinction, technology, and the ethics of conservation

What should the guiding ideals of conservation be in a new age of biotechnology?

Is extinction forever? Efforts are under way to use gene editing and other tools of biotechnology to "recreate" extinct species such as the woolly mammoth and the passenger pigeon. Could such "de-extinction" initiatives aid conservation by reviving species lost to habitat destruction and climate change? Or are they more likely to hinder conservation? What should the guiding ideals of conservation be in a new age of biotechnology? These are some of the questions addressed in Recreating the Wild: De-extinction, Technology, and the Ethics of Conservation, a new special report of the Hastings Center Report.

The report was edited by Gregory Kaebnick, a Hastings Center research scholar and editor of the Hastings Center Report, and Bruce Jennings, a senior advisor at the Center. The report grew out of a research project on de-extinction, led by Kaebnick and Jennings, that was part of a two-year collaboration of The Hastings Center and the Center for Humans and Nature, where Jennings is a senior scholar.

In their introduction, Kaebnick and Jennings observe that "we are living in what is widely considered the sixth major extinction," caused mainly by human activity. New biotechnology appears to offer the promise that "human ingenuity, a contributing factor in the extinction crisis, might achieve ... 'de-extinction'--in at least some cases, and with sometimes significant qualifications about whether the original species had been 'recreated' and whether it could resume its original place in the environment."

Major questions addressed in the special report include the following:

Is true de-extinction possible?

Advances in biology have revealed the ways the environment influences species' genomes. Even if scientists could produce creatures with DNA identical to that of extinct species, different environmental pressures would alter their genomes in novel ways, raising the possibility that those creatures would differ from the extinct species. "Species are entangled with other species, the land, and ecological events and processes," writes Ronald Sandler, director of the Ethics Institute at Northwestern University. "If scientists merely create organisms genetically similar to previously existing species, neither the species nor its relationships are regenerated." Still, some experts think that creating organisms that are similar to extinct species might have ecological benefits.

Does de-extinction support or undermine the goals of conservation?

Many scientists believe that although the maintenance of biodiversity benefits ecosystems, changes to the environment could make the reintroduction of extinct species difficult--possibly even ecologically disruptive. Curt Meine, a senior fellow with the Center for Humans and Nature and the Aldo Leopold Foundation, writes that species reintroduction does not take place in a "social or ecological vacuum" and that the interactions of a species with its physical and social environment are critical for its success.

Several commentators in the report raise the concern that the notion that extinct species might be "brought back" could weaken efforts to prevent extinctions. "By proposing that we can revive species through

modern technology, we give the impression that species are 'throwaway' items," write Robert DeSalle, a curator at the American Museum of Natural History's Sackler Institute for Comparative Genomics, and George Amato, director of the conservation genomics program at the institute. And Phil Seddon, chair of a recent International Union for Conservation of Nature task force that issued guidelines for attempting de-extinction, argues that, although conservationists need to be willing to use new biotechnologies for conservation goals, de-extinction may not be the best place to start.

What ideals should guide conservation as de-extinction and other biotechnological strategies become available?

Several essayists ask whether de-extinction goes too far in advancing human activity in the natural world. Christopher Preston, an ethicist at the University of Montana, argues that de-extinction is different from many other kinds of human activities because it tries to alter the deep structure of nature. Gregory Kaebnick asks whether de-extinction challenges the "gardening ethic" that some environmentalists have recently called for. He argues that the technologies show the need to think more carefully about what "good gardening" really means for a conservationist. In the version of gardening he defends, we should "think of nature as a place, a community--a threatened homeland," Kaebnick advises. "We live in it and dominate it, but we depend on it and cherish it. We should safeguard it."

http://bit.ly/2vcADQP

Primordial black holes may have helped to forge heavy elements

Astronomers like to say we are the byproducts of stars, stellar furnaces that long ago fused hydrogen and helium into the elements needed for life through the process of stellar nucleosynthesis.

As the late Carl Sagan once put it: "The nitrogen in our DNA, the calcium in our teeth, the iron in our blood, the carbon in our apple pies were made in the interiors of collapsing stars. We are made of star stuff."

But what about the heavier elements in the periodic chart, elements such as gold, platinum and uranium?

Astronomers believe most of these "r-process elements"--elements much heavier than iron--were created, either in the aftermath of the collapse of massive stars and the associated supernova explosions, or in the merging of binary neutron star systems.

"A different kind of furnace was needed to forge gold, platinum, uranium and most other elements heavier than iron," explained George Fuller, a theoretical astrophysicist and professor of physics who directs UC San Diego's Center for Astrophysics and Space Sciences. "These elements most likely formed in an environment rich with neutrons."

In a paper published August 7 in the journal Physical Review Letters, he and two other theoretical astrophysicists at UCLA--Alex Kusenko and Volodymyr Takhistov--offer another means by which stars could have produced these heavy elements: tiny black holes that came into contact with and are captured by neutron stars, and then destroy them.

Neutron stars are the smallest and densest stars known to exist, so dense that a spoonful of their surface has an equivalent mass of three billion tons.

Tiny black holes are more speculative, but many astronomers believe they could be a byproduct of the Big Bang and that they could now make up some fraction of the "dark matter"--the unseen, nearly non-interacting stuff that observations reveal exists in the universe.

If these tiny black holes follow the distribution of dark matter in space and co-exist with neutron stars, Fuller and his colleagues contend in their paper that some interesting physics would occur.

They calculate that, in rare instances, a neutron star will capture such a black hole and then devoured from the inside out by it.

This violent process can lead to the ejection of some of the dense neutron star matter into space.

"Small black holes produced in the Big Bang can invade a neutron star and eat it from the inside," Fuller explained.

"In the last milliseconds of the neutron star's demise, the amount of ejected neutron-rich material is sufficient to explain the observed abundances of heavy elements."

"As the neutron stars are devoured," he added, "they spin up and eject cold neutron matter, which decompresses, heats up and make these elements."

This process of creating the periodic table's heaviest elements would also provide explanations for a number of other unresolved puzzles in the universe and within our own Milky Way galaxy.

"Since these events happen rarely, one can understand why only one in ten dwarf galaxies is enriched with heavy elements," said Fuller. "The systematic destruction of neutron stars by primordial black holes is consistent with the paucity of neutron stars in the galactic center and in dwarf galaxies, where the density of black holes should be very high."

In addition, the scientists calculated that ejection of nuclear matter from the tiny black holes devouring neutron stars would produce three other unexplained phenomenon observed by astronomers.

"They are a distinctive display of infrared light (sometimes termed a "kilonova"), a radio emission that may explain the mysterious Fast Radio Bursts from unknown sources deep in the cosmos, and the positrons detected in the galactic center by X-ray observations," said Fuller.

"Each of these represent long-standing mysteries. It is indeed surprising that the solutions of these seemingly unrelated phenomena may be connected with the violent end of neutron stars at the hands of tiny black holes."

Funding for this project was provided by the National Science Foundation (PHY-1614864) at UC San Diego and by the U.S. Department of Energy (DE-SC0009937) at UCLA. Alex Kusenko was also supported, in part, by the World Premier International Research Center Initiative (WPI), MEXT, Japan.

http://nyti.ms/2vddb5W

Cleaning a Dirty Sponge Only Helps Its Worst Bacteria, Study Says

Microwaving your dirty sponge will only kill some of the bacteria on it, leaving the strongest, smelliest and potentially most pathogenic strains.

By JOANNA KLEIN AUG. 4, 2017

Stop. Drop the sponge and step away from the microwave.

That squishy cleaning apparatus is a microscopic universe, teeming with countless bacteria.

Some people may think that microwaving a sponge kills its tiny residents, but they are only partly right. It may nuke the weak ones, but the strongest, smelliest and potentially pathogenic bacteria will survive.

Then, they will reproduce and occupy the vacant real estate of the dead. And your sponge will just be stinkier and nastier and you may come to regret having not just tossed it, suggests a study published last month in Scientific Reports.

Bacteria are everywhere, so it's no surprise that a kitchen sponge would be full of them. But previous research had underestimated a sponge's quantity and range of bacteria.

By looking at the DNA and RNA in samples from 14 used sponges that may be as dirty as the one sitting in your sink right now, Markus Egert, a microbiologist at the University of Furtwangen in Germany, and his team identified 362 different species of bacteria living within them.

And the scientists were surprised to find how densely microbes occupied such close quarters: About 82 billion bacteria were living in just a cubic inch of space.

"That's the same density of bacteria you can find in human stool samples," Dr. Egert said. "There are probably no other places on earth with such high bacterial densities."

The sponge attracts bacteria — which arrive via food, the skin or other surfaces — with the perfect living conditions. There is lots of warm, wet and nutrient-rich space for them to thrive.

And among those taking advantage of these amenities, the scientists found, was a microbe called Moraxella osloensis. It is widespread in nature and lives on the human skin. It can cause infections in people with weak immune systems, although the risk posed by the bacteria found in sponges is hard to assess.

Moraxella osloensis is primarily responsible for the stench of dirty laundry, and it may also be the reason that your sponge eventually emits a funky odor.

The odor is a compound produced by the bacterium's metabolism. It eats fat. It excretes fat. And that fatty excrement stinks.

The thrifty among us may try to clean a sponge that starts to stink, but it's probably time to let it go. Disinfecting it, as many have tried, does not necessarily work.

You can microwave a sponge, throw it in the laundry or dishwasher, douse it in vinegar or other cleansing solutions or even cook it in a pot. But the researchers discovered more of the potentially pathogenic bacteria, like Moraxella osloensis, on the sponges collected from people who said they routinely disinfected them.

"When people at home try to clean their sponges, they make it worse," Dr. Egert said — similar to how people can encourage antibiotic resistant bacteria if they don't follow the doctor's orders. He says if you can't clean it perfectly, it may be best to replace it with a new one every week or so — especially "if it starts to move."

But if you would rather not create that much waste, run it through a laundry machine at the hottest setting using a powder detergent and bleach and then use it somewhere other than the kitchen that is less hygiene-sensitive, like the bathroom.

"Now I'm an expert in how to clean sponges," said Dr. Egert, who wants to compare disinfection methods in a follow-up study. "I'm waiting for the sponge industry to call me."

http://nyti.ms/2vcUn6R

Yellow Fever Outbreak That Threatened Brazil's Megacities Ends

After a fivefold rise in suspected cases of the disease in January, the peak of the outbreak, there were no new cases in July

By DONALD G. McNEIL Jr. AUG. 4, 2017

An alarming outbreak of yellow fever that threatened Brazil early this year appears to be over, according to data released this week by the Pan American Health Organization.

There were no new cases reported in Brazil in the last month, said officials of the agency, a regional branch of the World Health Organization. Of the neighboring countries to which the outbreak had spread, only Bolivia reported a case.

Concerns were first raised in January, when Brazil reported 712 suspected cases — a fivefold increase over normal levels. Most were in Minas Gerais, a rural state. About 40 of the state's residents died, and the governor declared a state of emergency.

Over the next few months, the outbreak spread, even reaching the states that are home to the megacities Rio de Janeiro and São Paulo. Some panicked residents reacted by killing monkeys, mistakenly blaming them for the spread.

The government distributed 20 million doses of yellow fever vaccine, including more than 3 million from the W.H.O. emergency stockpile.

In retrospect, reports show that total cases peaked in January, midsummer in the Southern Hemisphere, then fell substantially by March and nearly disappeared by June.

Yellow fever normally circulates in forest monkeys and forest mosquitoes, and it occasionally kills people who live or work there, such as loggers, miners and small farmers.

Scientists were particularly worried that infected humans would carry the virus into cities, where it could be picked up by Aedes aegypti mosquitoes, which thrive in huge numbers in urban slums and are aggressive biters of humans.

Had that happened, experts said, there was a serious risk that it would spread to the Northern Hemisphere, where vaccine for yellow fever is in short supply, or to Asia, where hundreds of millions of people with no natural immunity or history of vaccination live in tropical latitudes favored by Aedes aegypti mosquitoes.

http://bit.ly/2fleUCf

With genetic morph, a weird type of anthrax has emerged—and it's on a rampage

It's killing wildlife in African rainforests and may wipe out some chimp populations.

Beth Mole - 8/5/2017, 10:00 PM

After getting ahold of the genetic blueprints for molecular weapons, relatively harmless bacteria transformed into one that can cause anthrax—in places and animals where the original anthrax bacteria doesn't. And it's wreaking havoc.

Using data collected over a 26-year period, researchers found that this strange version of anthrax is running rampant in tropical rainforest habitats of Sub-Saharan Africa, killing off broad swaths of mammals. In fact, researchers estimated this week in *Nature* that this "rainforest anthrax" could wipe out chimpanzee populations in the Côte d'Ivoire's Taï National Park within the next 150 years. It's currently associated with nearly 40 percent of all chimp deaths there. And researchers are just getting started on understanding risks to humans, which have so far been thought to be low.

Among the living

Figuring out the scale and prevalence of this rainforest anthrax will be "critical for mitigating against the detrimental effects" to wildlife and "assessing human infection risk," the researchers, led by infectious disease expert Fabien Leendertz of the Robert Koch Institute in Germany, conclude.

Researchers have known about the existence of this alternative cause of anthrax for more than a decade. However, they've known little about its whereabouts and spread.

Classic anthrax is caused by *Bacillus anthracis*, which tends to strike ungulates (hoofed mammals) in seasonal outbreaks in arid locales, such as the African savannahs. The bacteria can cause infection in skin, lungs, or intestines. In humans, *B. anthracis* causes ghastly skin lesions, and severe respiratory and intestinal infections—which have mortality rates as high as 85 and 60 percent, respectively.

The alternative anthrax bacteria appear to cause an identical anthrax disease in animal models. But, those bacteria aren't *B. anthracis*; they're cousins, *B. cereus*, commonly found in soil and food. Usually, these are relatively harmless, with some strains known to cause a minor fraction of food poisoning cases. But the ones causing alternative anthrax are different. They just so happen to have gotten their grips on *B. anthracis*' virulence plasmids—circular, shareable bits of DNA that contain the genetic code for their disease-causing gene products.

Researchers dubbed these alternative anthrax bacteria: *B. cereus* biovar *anthracis*, or Bcbva.

Spreading the disease

Researchers saw Bcbva in Taï National Park (TNP) in 2001. With carcasses piling up, Leendertz and colleagues started testing them in 2004—sampling 204 in total—as well as bones of 75 mammals, collected since 1989. In 2008, they started testing carrion flies, which can spread Bcbva, netting 1,634 fly samples. To assess Bcbva prevalence, they also gathered 1,089 fly samples and 136 bones from 16 sites in 11 other sub-Saharan countries.

From the TNP samples, they wound up with 178 whole genome-sequences of Bcbva from 80 of the carcasses (40 percent), 26 bones (35 percent of bone samples), and 80 flies (five percent). They found that the killer microbes were genetically diverse, suggesting that they had been active and spreading in the region for a while.

Regional sampling suggested Bcbva was widespread and indiscriminate. It showed up in 5 of 11 test locations and seemed to be present all the time, not just in seasonal bouts. It also didn't just strike

ungulates but a variety of animals: chimps, mongooses, porcupines, six monkey species, and duikers (a type of antelope).

But the chimps seemed to be hit particularly hard by the microbe. Based on population data and modeling on the already endangered chimp populations studied in TNP, the researchers estimate a high likelihood that Bcbva will wipe out these slow-reproducing primates within the next 150 years.

In an accompanying commentary, senior biology editor and ecologist Anna Armstrong noted that the dim projection doesn't account for other problems. The risk "is only set to increase if chimp mortality from hunting and human-borne diseases continues to rise."

Nature, *2017*. DOI: 10.1038/nature23309 (About DOIs).

http://bit.ly/2vwdYRZ

'Loneliness epidemic' set to become a public health crisis

New research confirms the deadly effects of increasing social isolation.

Multiple recent studies have found that loneliness is set to reach epidemic proportions through the Western world by 2030. Now, one of the most prominent researchers in the field suggests that it will soon outstrip obesity as a public health crisis.

Julianne Holt-Lunstad of Brigham Young University in Utah, US, prepared new research to present to the 125th Annual Convention of the American Psychological Association, held in Washington DC this month.

The work was based on a combination of two meta-analyses. The first involved 148 studies covering 300,000 people, and found that social connectedness to family or community reduced the risk of all-cause mortality by 50%.

The analysis also incorporated a further 70 studies covering 3.4 million people from North America, Australia and Europe. These studies looked at the health outcomes of people subject to social isolation, loneliness or living alone. They concluded that all three conditions – individually or in combination – increased the risk of premature death. The risk escalation was in each study either equal to or greater than the risk posed by obesity.

"Being connected to others socially is widely considered a fundamental human need," says Holt-Lunstad. "It is crucial to both well-being and survival. Extreme examples show infants in custodial care who lack human contact fail to thrive and often die, and indeed, social isolation or solitary confinement has been used as a form of punishment."

In 2015 Holt-Lunstad published a pioneering study into the health effects of loneliness. That study, in the journal *Perspectives on Psychological Science*, was also a meta-analysis, found that social isolation, loneliness or living alone increased the risk of premature death by, respectively, 29%, 26%, and 32%.

The study helped to change the focus from a lot of earlier research, which had tended to see loneliness as the product of some other condition, such as depression, or poverty.

One of the primary challenges for researchers in the field is that loneliness and social isolation have high individualistic expressions, so a rigorous definition of the condition is difficult. Attempts to define it by using a checklist approach have produced, often, distorted results.

"Loneliness is a subjective experience," said social work professor Dr Mark Hughes, from Southern Cross University in Queensland, Australia in an interview earlier this year.

"It's the feeling that you don't have sufficient social connections. It's an internal, emotional response to your life situation. I think measures will always be based on self-reporting of the experience of loneliness."

Levels and degrees of experience are thus elusive, although the broad picture – that loneliness is increasing, and its public health implications are serious – are largely undisputed.

In 2016, the Australian crisis help organisation Lifeline released the results of its own survey into loneliness. It reported that as many as 80% of respondents said they had experienced significant periods of loneliness.

Perhaps significantly, 60% of respondents who said they "often" felt lonely lived with a partner, and many of them with children.

This apparently counter-intuitive finding accords with research published in 2009 which found levels of loneliness were higher in societies that were traditionally family-orientated compared to those where individualism and independence were seen as desirable virtues.

Although results vary, most studies into the public health implications of loneliness have reported that the risk of isolation increases with age. Holt-Lunstad warns that older people continue to constitute a growing proportion of the population, the situation is set to worsen.

"With an increasing aging population, the effect on public health is only anticipated to increase," she says. "Indeed, many nations around the world now suggest we are facing a 'loneliness epidemic'. The challenge we face now is what can be done about it."

http://bit.ly/2flvmCE

Gold specks raise hopes for better cancer treatments

A tiny medical device containing gold specks could boost the effects of cancer medication and reduce its harm, research suggests.

Scientists have completed a study which showed that gold increased the effectiveness of drugs used to treat lung cancer cells.

Experts say that the findings could help researchers use the device to reduce side effects of current chemotherapies by precisely targeting diseased cells without damaging healthy tissue.

Gold is a safe chemical element and has the ability to accelerate - or catalyse - chemical reactions.

Researchers at the University of Edinburgh discovered properties of the precious metal that allow these catalytic abilities to be accessed in living things without any side effects.

Minute fragments, known as gold nanoparticles, were encased in a chemical device by the research team to control these highly-specific reactions in exact locations.

The device was shown to catalyse a directed chemical reaction when implanted in the brain of zebrafish, suggesting it can be used in living animals.

Gold nanoparticles also activated anti-cancer medicines that had been applied to lung cancer cells in a dish, increasing the drugs' effectiveness.

Some 450 people die from cancer every day in the UK. A cancer diagnosis is made every two minutes. Medications are improving, but often damage healthy cells.

The study was carried out in collaboration with researchers at the University of Zaragoza's Institute of Nanoscience of Aragon in Spain. It was part-funded by Cancer Research UK (CRUK), and the Engineering and Physical Sciences Research Council and is published in the journal Angewandte Chemie.

Dr Asier Unciti-Broceta from the University of Edinburgh's CRUK Edinburgh Centre, said: "We have discovered new properties of gold that were previously unknown and our findings suggest that the metal could be used to release drugs inside tumours very safely.

"There is still work to do before we can use this on patients, but this study is a step forward. We hope that a similar device in humans could one day be implanted by surgeons to activate chemotherapy directly in tumours and reduce harmful effects to healthy organs."

Dr Áine McCarthy, Cancer Research UK's senior science information officer said: "By developing new, better ways of delivering cancer drugs, studies like this have the potential to improve cancer treatment and reduce side effects. In particular, it could help improve treatment for brain tumours and other hard-to-treat cancers. The next steps will be to see if this method is safe to use in people, what its long- and short-term side effects are, and if it's a better way to treat some cancers."