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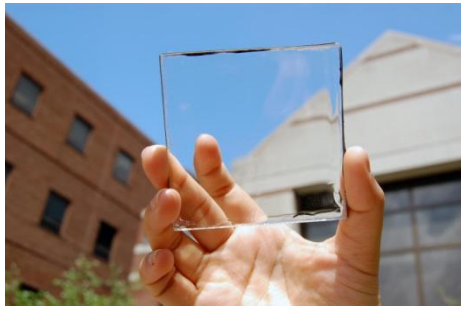
Transparent solar technology represents 'wave of the future'

Highly transparent solar cells represent the wave of the future for new solar applications



EAST LANSING, Mich. - See-through solar materials that can be applied to windows represent a massive source of untapped energy and could harvest as much power as bigger, bulkier rooftop solar units, scientists report today in Nature Energy.

See-through solar-harvesting applications, such as this module pioneered at Michigan State University, could potentially produce 40 percent of U.S. electricity demand. **Michigan State University**

Led by engineering researchers at Michigan State University, the authors argue that widespread use of such highly transparent solar applications, together with the rooftop units, could nearly meet U.S. electricity demand and drastically reduce the use of fossil fuels.

"Highly transparent solar cells represent the wave of the future for new solar applications," said Richard Lunt, the Johansen Crosby Endowed Associate Professor of Chemical Engineering and Materials Science at MSU.

"We analyzed their potential and show that by harvesting only invisible light, these devices can provide a similar electricity-generation potential as rooftop solar while providing additional functionality to enhance the efficiency of buildings, automobiles and mobile electronics."

Lunt and colleagues at MSU pioneered the development of a transparent luminescent solar concentrator that when placed on a window creates solar energy without disrupting the view. The thin, plastic-like material can be used on buildings, car windows, cell phones or other devices with a clear surface.

The solar-harvesting system uses organic molecules developed by Lunt and his team to absorb invisible wavelengths of sunlight. The researchers can "tune" these materials to pick up just the ultraviolet and the near-infrared wavelengths that then convert this energy into electricity.

Moving global energy consumption away from fossil fuels will require such innovative and cost-effective renewable energy technologies. Only about 1.5 percent of electricity demand in the United States and globally is produced by solar power.

But in terms of overall electricity potential, the authors note that there is an estimated 5 billion to 7 billion square meters of glass surface in the United States. And with that much glass to cover, transparent solar technologies have the potential of supplying some 40 percent of energy demand in the U.S. - about the same potential as rooftop solar units.

"The complimentary deployment of both technologies," Lunt said, "could get us close to 100 percent of our demand if we also improve energy storage."

Lunt said highly transparent solar applications are recording efficiencies above 5 percent, while traditional solar panels typically are about 15 percent to 18 percent efficient. Although transparent solar technologies will never be more efficient at converting solar energy to electricity than their opaque counterparts, they can get close and offer the potential to be applied to a lot more additional surface area, he said.

Right now, transparent solar technologies are only at about a third of their realistic overall potential, Lunt added.

"That is what we are working towards," he said. "Traditional solar applications have been actively researched for over five decades, yet we have only been working on these highly transparent solar cells for about five years. Ultimately, this technology offers a promising route to inexpensive, widespread solar adoption on small and large surfaces that were previously inaccessible."

The work is funded by the National Science Foundation and the U.S. Department of Education.

Lunt's coauthors are Christopher Traverse, a doctoral student in engineering at MSU, and Richa Pandey and Miles Barr with Ubiquitous Energy Inc., a company Lunt cofounded with Barr to commercialize transparent solar technologies.

http://bit.ly/2qJDj1H

Why Does Our Universe Have 3 Dimensions?

We take for granted that we live in a world of three dimensions governed by the laws of physics, and don't often wonder why.

By Nancy Atkinson, Seeker | October 23, 2017 02:48pm ET

But a group of physicists just hatched a new theory that they think may explain our three-dimensional universe.

The physicists think that their new model could also explain inflation, the exponential expansion of space the universe experienced just moments after the Big Bang.

Thomas Kephart from Vanderbilt University and four of his colleagues from around the world wanted to figure out why our universe seemingly has just three dimensions, especially since, as they wrote, "quantum gravity scenarios such as string theory... assume nine or ten space dimensions at the fundamental level."

They combined particles physics with mathematical knot theory to try and work this out, borrowing the concept of "flux tubes," which are flexible strands of energy that link elementary particles together.

Quarks, the elementary particles that make up protons and neutrons, are held together by another type of elementary particle called a gluon that "glues" quarks together. Gluons bond positive quarks to matching negative antiquarks with these flux tube energy strands.

Normally, the flux tube that links a quark and antiquark would disappear when the two particles come into contact — they would self-annihilate. But, the team said in a paper published by the European Physical Journal C, if two or more flux tubes become intertwined, it becomes stable. If the tubes take the form of a knot, they become even more stable and can outlive the particles that created it.

"A knot or link between two flux tubes is only classically stable if these are unable to intersect and either reconnect or pass through each other," the researchers wrote.

"Such intercommutations lead to the well-known scaling behavior in cosmic string networks, which has been observed in several examples of non-interacting strings."

In moments of transition, such as what happened during the Big Bang, the linked particles would get pulled apart, and the flux tube would get longer until it reaches a point where it breaks.

When it does, it releases enough energy to form a second quark-antiquark pair that splits up and binds with the original particles, producing two pairs of bound particles.

The physicists equated this to how cutting a bar magnet in half produces two smaller magnets that both have north and south poles.

If the tubes were knotted together, they could quickly expand and multiply. The team calculated the energy that this flux tube network might contain and found that it would be enough to power an early period of cosmic inflation.

While this sounds like an incredible amount of action to take place in such a short period of time — inflation theory suggests that the universe expanded exponentially in milliseconds — Kephart told Seeker that flux tubes form naturally during times of transition.

"Flux tubes form in phase transitions where complex forms of matter can arise," he explained in an email. "For example, water vapor is structurally simple, but if it is rapidly cooled you get a flurry of snowflakes — they all look different and the new phase seems much more complex."

In an environment of extremely high energy, the team said that the quark-gluon plasma would have been an ideal environment for rapid flux tube formation in the very early universe.

But, crucially, they noted that this would only work if the universe existed in three dimensions. If you add more dimensions, the process becomes unstable.

"Of all possible dimensionalities of space, our mechanism picks out three as the only number of dimensions that can inflate and thus become large," the team wrote. "This model may explain why we live in three large spatial dimensions, since knotted/linked tubes are topologically unstable in higher-dimensional space-times."

This would technically agree with a computer model from 2012 where Japanese scientists found that at the moment of the Big Bang, the universe had 10 dimensions, but only three of these spatial dimensions expanded. So, the three-dimensional space we experience could have formed from 10 dimensions, just as superstring theory predicts.

Their new theory would also agree with certain gauge theories, which are theories used by physicists that describe the limits of physical laws and how they apply to symmetric transformations.

Kephart noted that this new flux tube theory also encompasses what happened after inflation.

"Not only does our flux tube network provide the energy needed to drive inflation, it also explains why it stopped so abruptly," he said in a statement. "As the universe began expanding, the flux-tube network began decaying and eventually broke apart, eliminating the energy source that was powering the expansion."

The researchers say that when the network broke down, it filled the universe with a gas of subatomic particles and radiation, allowing the evolution of the universe to continue to what we see today.

"This combines knowledge of gauge theories and the possibility that an initial uniform configuration can condense into flux tubes," Kephart told Seeker, "along with the fact that knots and links for strings can only be stable in 3D, plus the current state of the theory of the early Universe and the need for a natural way to inflate."

While this is all theoretical, Kephart said that the next step would be to continuing to develop their theory until it can make some predictions about the nature of the universe that can actually be tested.

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Crops evolving 10 millennia before experts thought

Ancient peoples began to systematically affect evolution of crops up to 30,000 years ago - ten millennia before experts previously thought, says new University of Warwick research

Ancient hunter-gatherers began to systemically affect the evolution of crops up to thirty thousand years ago - around ten millennia before experts previously thought - according to new research by the University of Warwick.

Professor Robin Allaby, in Warwick's School of Life Sciences, has discovered that human crop gathering was so extensive, as long ago as the last Ice Age, that it started to have an effect on the evolution of rice, wheat and barley - triggering the process which turned these plants from wild to domesticated.

In Tell Qaramel, an area of modern day northern Syria, the research demonstrates evidence of einkorn being affected up to thirty thousand years ago, and rice has been shown to be affected more than thirteen thousand years ago in South, East and South-East Asia.

Furthermore, emmer wheat is proved to have been affected twenty-five thousand years ago in the Southern Levant - and barley in the same geographical region over twenty-one thousand years ago.

The researchers traced the timeline of crop evolution in these areas by analysing the evolving gene frequencies of archaeologically uncovered plant remains.

Wild plants contain a gene which enables them to spread or shatter their seeds widely. When a plant begins to be gathered on a large scale, human activity alters its evolution, changing this gene and causing the plant to retain its seeds instead of spreading them - thus adapting it to the human environment, and eventually agriculture.

Professor Allaby and his colleagues made calculations from archaeobotanical remains of crops mentioned above that contained 'non-shattering' genes - the genes which caused them to retain their

seeds - and found that human gathering had already started to alter their evolution millennia before previously accepted dates.

The study shows that crop plants adapted to domestication exponentially around eight thousand years ago, with the emergence of sickle farming technology, but also that selection changed over time. It pinpoints the origins of the selective pressures leading to crop domestication much earlier, and in geological eras considered inhospitable to farming.

Demonstrating that crops were being gathered to the extent of being pushed towards domestication up to thirty thousand years ago proves the existence of dense populations of people at this time.

Professor Robin Allaby commented:

"This study changes the nature of the debate about the origins of agriculture, showing that very long term natural processes seem to lead to domestication - putting us on a par with the natural world, where we have species like ants that have domesticated fungi, for instance."

The research, 'Geographic mosaics and changing rates of cereal domestication', is published in Philosophical Transactions of the Royal Society B.

http://bit.ly/2qIT9Kq

Patients prefer doctors not use computers in exam room

A new study suggests that people with advanced cancer prefer doctors communicate with them face-to-face with just a notepad in hand rather than repeatedly using a computer.

ALEXANDRIA, Va. - These findings will be presented at the upcoming 2017 Palliative and Supportive Care in Oncology Symposium in San Diego, California.

"To our knowledge, this is the only study that compares exam room interactions between people with advanced cancer and their physicians, with or without a computer present," said lead study author Ali Haider, MD, an Assistant Professor at the University of Texas MD Anderson Cancer Center in the Department of Palliative, Rehabilitation, and Integrative Medicine, which also funded the study.

Many doctors now use a computer software program for managing electronic health records. The researchers were concerned that it might impair communication with patients and also knew from earlier research that people with chronic health concerns, and often accompanying emotional issues, want their doctors to talk to them directly.

About the Study

The researchers filmed four approximately 2-minute videos that featured actors who were carefully scripted and used the same gestures, expressions, and other nonverbal communication in each video to minimize bias:

Video 1: Doctor A in a face-to-face consultation with just a notepad in hand

Video 2: Doctor A in a consultation using a computer

Video 3: Doctor B in a face-to-face consultation with just a notepad in hand

Video 4: Doctor B in a consultation using a computer

The patients in the study had either localized, recurrent, or metastatic disease. Ninety percent were fully physically functional, and all were English speakers. To further standardize and control their assessment, the researchers captured patient information on psychosocial factors, age, and level of education upon enrollment.

The researchers randomly assigned 120 patients to four equal-sized groups. After viewing their first video, the patients completed a validated questionnaire rating the doctor's communication skills, professionalism, and compassion. Subsequently, each group was assigned to a video topic (face-to-face or computer) they had not viewed previously featuring an actor-doctor they had not viewed in the first video. A follow-up questionnaire was given after this round of viewing, and the patients were also asked to rate their overall physician preference.

Key Findings

After the first round of viewing, the patients rated doctors in the face-to-face video as having more compassion and better communication skills and professionalism than the doctors who used the computer in

the exam room. After having watched both videos, 72% of participants favored the face-to-face interaction.

"We know that having a good rapport with patients can be extremely beneficial for their health," said Dr. Haider. "Patients with advanced disease need the cues that come with direct interaction to help them along with their care." The researchers note that their study answers questions about patients' perceptions, but not how to address the issue of computer use in an exam room.

Next Steps

"Our study was done at an outpatient clinic, so it is probably more pertinent in that setting compared to a hospital where patient-doctor interactions are more frequent and rigorous," said Dr. Haider. "We are pretty certain that people will permit another entity in the exam room, but our study shows that if the third entity is a computer, the computer is not preferred."

The researchers believe that they would probably find the same results if the study was conducted with people with early-stage cancer. However, they weren't so sure about a younger population with higher computer literacy and said that population might be the subject of a future study.

http://bit.ly/2hfXncN

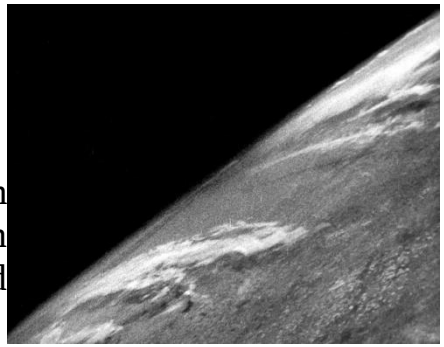
The first photograph of Earth taken from space

On 24 October 1946, rocket scientists captured the first images of Earth taken from space.

This is the first photograph of Earth ever taken from space. It was captured on 24 October 1946 from a rocket 105 km above the ground that had been launched from the White Sands Missile Range in New Mexico, USA.

The rocket was a German V2, captured by the Americans at the end of World War II. Hundreds of scientists and engineers from the Nazi rocket program were vital to the postwar development of the American and Russian space programs.

Though the V2 had rained terror on London and other cities during the war, in peacetime the explosive warhead was removed and replaced with a package of scientific instruments. These included a 35mm motion-picture camera set to snap one picture every second and a half.



The first photograph of Earth from space, taken on 24 October 1946.
White Sands Missile Range / Applied Physics Laboratory

The resulting images, developed from film dropped back to Earth in a tough steel canister, were like nothing that had been seen before.

Until this point, the highest vantage point from which photos had been taken was some 22 km, aboard a high-altitude balloon.

The balloon pictures had shown the curvature of the Earth at the horizon, but the rocket photos opened new possibilities. Clyde Holliday, the engineer who developed the camera, saw the potential: in a 1950 *National Geographic* article, he predicted that one day “the entire land area of the globe might be mapped in this way”.

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MIT neuroscientists build case for new theory of memory formation

Existence of "silent engrams" suggests that existing models of memory formation should be revised.

CAMBRIDGE, MA -- Learning and memory are generally thought to be composed of three major steps: encoding events into the brain network, storing the encoded information, and later retrieving it for recall.

Two years ago, MIT neuroscientists discovered that under certain types of retrograde amnesia, memories of a particular event could be stored in the brain even though they could not be retrieved through natural recall cues. This phenomenon suggests that existing models of memory formation need to be revised, as the researchers propose in a

new paper in which they further detail how these "silent engrams" are formed and re-activated.

The researchers believe their findings offer evidence that memory storage does not rely on the strengthening of connections, or "synapses," between memory cells, as has long been thought. Instead, a pattern of connections that form between these cells during the first few minutes after an event occurs are sufficient to store a memory.

"One of our main conclusions in this study is that a specific memory is stored in a specific pattern of connectivity between engram cell ensembles that lie along an anatomical pathway. This conclusion is provocative because the dogma has been that a memory is instead stored by synaptic strength," says Susumu Tonegawa, the Picower Professor of Biology and Neuroscience, the director of the RIKEN-MIT Center for Neural Circuit Genetics at the Picower Institute for Learning and Memory, and the study's senior author.

The researchers also showed that even though memories held by silent engrams cannot be naturally recalled, the memories persist for at least a week and can be "awakened" days later by treating cells with a protein that stimulates synapse formation.

Dheeraj Roy, a recent MIT PhD recipient, is the lead author of the paper, which appears in the Proceedings of the National Academy of Sciences the week of Oct. 23. Other authors are MIT postdoc Shruti Muralidhar and technical associate Lillian Smith.

Silent memories

Neuroscientists have long believed that memories of events are stored when synaptic connections, which allow neurons to communicate with each other, are strengthened. Previous studies have found that if synthesis of certain cellular proteins is blocked in mice immediately after an event occurs, the mice will have no long-term memory of the event.

However, in a 2015 paper, Tonegawa and his colleagues showed for the first time that memories could be stored even when synthesis of the cellular proteins is blocked. They found that while the mice could not recall those memories in response to natural cues, such as being placed in the cage where a fearful event took place, the memories were still there and could be artificially retrieved using a technique known as optogenetics.

The researchers have dubbed these memory cells "silent engrams," and they have since found that these engrams can also be formed in other situations. In a study of mice with symptoms that mimic early Alzheimer's disease, the researchers found that while the mice had trouble recalling memories, those memories still existed and could be optogenetically retrieved.

In a more recent study of a process called systems consolidation of memory, the researchers found engrams in the hippocampus and the prefrontal cortex that encoded the same memory. However, the prefrontal cortex engrams were silent for about two weeks after the memory was initially encoded, while the hippocampal engrams were active right away. Over time, the memory in the prefrontal cortex became active, while the hippocampal engram slowly became silent.

In their new PNAS study, the researchers investigated further how these silent engrams are formed, how long they last, and how they can be re-activated.

Similar to their original 2015 study, they trained mice to fear being placed in a certain cage, by delivering a mild foot shock. After this training, the mice freeze when placed back in that cage. As the mice were trained, their memory cells were labeled with a light-sensitive protein that allows the cells to be re-activated with light. The researchers also inhibited the synthesis of cellular proteins immediately after the training occurred.

They found that after the training, the mice did not react when placed back in the cage where the training took place. However, the mice did freeze when the memory cells were activated with laser light while the animals were in a cage that should not have had any fearful associations. These silent memories could be activated by laser light for up to eight days after the original training.

Making connections

The findings offer support for Tonegawa's new hypothesis that the strengthening of synaptic connections, while necessary for a memory to be initially encoded, is not necessary for its subsequent long-term storage. Instead, he proposes that memories are stored in the specific pattern of connections formed between engram cell ensembles. These connections, which form very rapidly during encoding, are distinct from the synaptic strengthening that occurs later (within a few hours of the event) with the help of protein synthesis.

"What we are saying is that even without new cellular protein synthesis, once a new connection is made, or a pre-existing connection is strengthened during encoding, that new pattern of connections is maintained," Tonegawa says. "Even if you cannot induce natural memory recall, the memory information is still there."

This raised a question about the purpose of the post-encoding protein synthesis. Considering that silent engrams are not retrieved by natural cues, the researchers believe the primary purpose of the protein synthesis is to enable natural recall cues to do their job efficiently.

The researchers also tried to reactivate the silent engrams by treating the mice with a protein called PAK1, which promotes the formation of synapses. They found that this treatment, given two days after the original event took place, was enough to grow new synapses between engram cells. A few days after the treatment, mice whose ability to recall the memory had been blocked initially would freeze after being placed in the cage where the training took place. Furthermore, their reaction was just as strong as that of mice whose memories had been formed with no interference.

Along with the researchers' previous findings on silent engrams in early Alzheimer's disease, this study suggests that re-activating certain synapses could help restore some memory recall function in patients with early stage Alzheimer's disease, Roy says.

The research was funded by the RIKEN Brain Science Institute, the Howard Hughes Medical Institute, and the JPB Foundation.

http://bit.ly/2iGCGKR

Blood-thinning drugs appear to protect against dementia as well as stroke in AF patients

Blood-thinning drugs reduce the risk of stroke and are also associated with significant reduction in the risk of dementia

Blood-thinning drugs not only reduce the risk of stroke in patients with atrial fibrillation (AF) but are also associated with a significant reduction in the risk of dementia, according to new research published today (Wednesday) in the European Heart Journal [1].

Among 444,106 patients with atrial fibrillation (an abnormal heart rhythm), those who were taking anticoagulant drugs to prevent blood clots at the start of the study had a 29% lower risk of developing dementia than patients who were not on anticoagulant treatment. When the researchers looked at what happened during the period of time that the patients continued to take the drugs, they found an even bigger, 48% reduction in the risk of dementia.

This is the largest study ever to examine the link between anticoagulant treatment and dementia in AF patients. It looked at data from Swedish registries for patients between 2006 and 2014, and, despite its retrospective nature, which means it cannot show causal effect, the researchers believe that the results strongly suggest that oral anticoagulants protect against dementia in AF patients.

"In order to prove this assumption, randomized placebo controlled trials would be needed, but... such studies cannot be done because of ethical reasons. It is not possible to give placebo to AF patients and then wait for dementia or stroke to occur," write Leif Friberg and Mårten Rosenqvist from the Karolinska Institute (Stockholm, Sweden) in their EHJ paper.

AF is known to carry an increased risk of stroke and dementia, and anticoagulants have been shown to reduce the likelihood of stroke. Until now it was not clear whether anticoagulants could also prevent dementia; however, it was thought possible because if the drugs can prevent the big blood clots that cause stroke, they might also protect

against the small clots that can cause unnoticed microscopic strokes that eventually lead to cognitive deterioration.

The researchers identified all patients in Sweden who had a diagnosis of AF between 2006-2014. They checked on what drugs had been prescribed and dispensed following the diagnosis. They followed the patients' progress and this provided them with 1.5 million years of follow-up, during which time 26,210 patients were diagnosed with dementia.

When they first joined the study, 54% of patients were not taking oral anticoagulants such as warfarin, apixaban, dabigatran, edoxaban or rivaroxaban. The researchers found that the strongest predictors for dementia were lack of oral anticoagulant treatment, aging, Parkinson's disease and alcohol abuse. They also found that the sooner oral anticoagulant treatment was started after a diagnosis of AF, the greater was the protective effect against dementia.

Dr Friberg, who is associate professor of cardiology at the Karolinska Institute, said the important implications from these findings were that patients should be started on oral anticoagulant drugs as soon as possible after diagnosis of AF and that they should continue to take the drugs.

"Doctors should not tell their patients to stop using oral anticoagulants without a really good reason. Explain to your patients how these drugs work and why they should use them. An informed patient who understands this is much more likely to comply and will be able to use the drugs safely and get better benefits. To patients I would say 'don't stop unless your doctor says so. Have your doctor explain why you should take the drug so that you feel you understand and agree'.

"Patients start on oral anticoagulation for stroke prevention but they stop after a few years at an alarmingly high rate. In the first year, approximately 15% stop taking the drugs, then approximately 10% each year. In this study we found that only 54% of patients were on oral anticoagulant treatment. If you know that AF eats away your brain at a slow but steady pace and that you can prevent it by staying on treatment, I think most AF patients would find this a very strong argument for continuing treatment.

"As a clinician I know there are AF patients who have a fatalistic view upon stroke. Either it happens or it does not. Few patients are fatalistic about dementia, which gradually makes you lose your mind. No brain can withstand a constant bombardment of microscopic clots in the long run. Patients probably want to hang on to as many of their little grey cells for as long as they can. In order to preserve what you´ve got, you should take care to use anticoagulants if you are diagnosed with AF, as they have been proved to protect against stroke and, which this study indicates, also appear to protect against dementia."

The study also found that there was no difference in dementia prevention between the older blood-thinning drug warfarin and the newer oral anticoagulants.

In addition to the fact that this study cannot prove or disprove a causal relationship between oral anticoagulants and dementia, some other limitations include the lack of complete medical histories for the patients, including details of other diseases, and the fact that dementia is insidious and is not necessarily diagnosed immediately, meaning that the true prevalence of dementia is probably higher than reported.

Notes:

[1] "Less dementia with oral anticoagulation in atrial fibrillation", by Leif Friberg and Mårten Rosenqvist. European Heart Journal. doi:10.1093/eurheartj/ehx579

http://bit.ly/2zVx9Em

Daydreaming is good. It means you're smart

Brain study suggests mind wandering at work and home may not be as bad as you might think

A new study from the Georgia Institute of Technology suggests that daydreaming during meetings isn't necessarily a bad thing. It might be a sign that you're really smart and creative.

"People with efficient brains may have too much brain capacity to stop their minds from wandering," said Eric Schumacher, the Georgia Tech associate psychology professor who co-authored the study.

Schumacher and his students and colleagues, including lead co-author Christine Godwin, measured the brain patterns of more than 100 people while they lay in an MRI machine. Participants were instructed to focus on a stationary fixation point for five minutes. The Georgia Tech team used the data to identify which parts of the brain worked in unison.

"The correlated brain regions gave us insight about which areas of the brain work together during an awake, resting state," said Godwin, a Georgia Tech psychology Ph.D. candidate.

"Interestingly, research has suggested that these same brain patterns measured during these states are related to different cognitive abilities."

Once they figured out how the brain works together at rest, the team compared the data with tests the participants that measured their intellectual and creative ability. Participants also filled out a questionnaire about how much their mind wandered in daily life.

Those who reported more frequent daydreaming scored higher on intellectual and creative ability and had more efficient brain systems measured in the MRI machine.

"People tend to think of mind wandering as something that is bad. You try to pay attention and you can't," said Schumacher. "Our data are consistent with the idea that this isn't always true. Some people have more efficient brains."

Schumacher says higher efficiency means more capacity to think, and the brain may mind wander when performing easy tasks.

How can you tell if your brain is efficient? One clue is that you can zone in and out of conversations or tasks when appropriate, then naturally tune back in without missing important points or steps.

"Our findings remind me of the absent-minded professor -- someone who's brilliant, but off in his or her own world, sometimes oblivious to their own surroundings," said Schumacher. "Or school children who are too intellectually advanced for their classes. While it may take five minutes for their friends to learn something new, they figure it out in a minute, then check out and start daydreaming."

Godwin and Schumacher think the findings open the door for follow-up research to further understand when mind wandering is harmful, and when it may actually be helpful.

"There are important individual differences to consider as well, such as a person's motivation or intent to stay focused on a particular task," said Godwin.

The paper, "Functional connectivity within and between intrinsic brain networks correlates with trait mind wandering," is published in the journal Neuropsychologia.

http://bbc.in/2iFDlvS

Daydreaming brain network used in autopilot

The part of the brain associated with daydreaming also allows us to perform tasks on autopilot, a study has found.

By Katie Silver Health reporter

A collection of brain regions known as the "default mode network" (DMN) is active when we are daydreaming or thinking about the past or future. Cambridge University researchers found it also allows us to switch to autopilot once we are familiar with a task, such as driving a familiar route. There is even hope the findings can help people with mental illness.

Previous research has found the DMN is more active during states of rest, and that it can behave abnormally in conditions such as Alzheimer's disease, schizophrenia and attention deficit hyperactivity disorder (ADHD). But researchers have remained unclear about its exact role.

Switching to manual

For the current study, 28 volunteers were asked to match a target card, such as the two of clubs, with one of four cards shown.

They had to work out if the cards were supposed to be matched on colour, number or shape through trial and error. Their brain activity was monitored throughout using a scanner.

While they were learning the rule, known as the acquisition stage, a part of the brain known as the dorsal attention network was more

active. It has been associated with processing information that demands attention. Once they knew the rule and were applying it, the DMN was more active. They were particularly good at the task if their DMN activity was associated with activity in the hippocampus, the part of the brain associated with memory.

Lead author Deniz Vatansever says the DMN allows us to predict what is going to happen and reduce our need to think. "It is essentially like an autopilot that helps us make fast decisions when we know what the rules of the environment are.

"So, for example, when you're driving to work in the morning along a familiar route, the default mode network will be active, enabling us to perform our task without having to invest lots of time and energy into every decision."

When the environment changes, and no longer conforms to our expectations, Dr Vatansever said our brain enters a "manual mode" that overrides the automatic system, or DMN activity.

The researchers hope their findings will help those with mental health disorders - such as addiction, depression and obsessive compulsive disorder who can have automatic thought patterns that drive repeated, unpleasant behaviour.

http://bit.ly/2yYxvvW

Heart failure therapy hope as drug blocks deadly muscle scarring

A potential treatment to prevent deadly muscle scarring that contributes to chronic heart failure has been uncovered by scientists.

The therapy could also prevent scarring of the skeletal muscles we use to move our arms and legs, a cause of long-term disabilities.

The treatment works by targeting molecules on the surface of scar-forming cells, called alpha V integrins.

A research team, led by the University of Edinburgh, found that blocking these molecules with a new experimental drug helped to reduce scarring following heart or skeletal muscle injury.

The treatment works even when the muscle scarring process has already started, the research with mice found.

The next step will be to test the drug in clinical trials with people to check whether it can help to reduce scarring in patients with chronic heart failure, and also patients with skeletal muscle scarring.

Scarring is a natural response to tissue injury, but in excess it can stop muscles from working effectively.

In many people excessive scarring - known as fibrosis - is permanent, and causes muscles to become stiff and less compliant.

When fibrosis occurs in cardiac muscle during chronic heart failure, the heart muscle is less able to contract properly and pump blood around the body.

The team found that scarring is initiated by cells around the lining of blood vessels, which have alpha V integrins on their surface.

Lab tests on human cells of this type - called mesenchymal cells - found that blocking alpha V integrins stops them from becoming activated and blocks the scarring process.

The study, published in Nature Communications, was supported by the Wellcome Trust and the British Heart Foundation. Funding was also received from the Royal College of Surgeons of Edinburgh.

Professor Neil Henderson, of the Medical Research Council Centre for Inflammation Research at the University of Edinburgh, said: "Cardiac fibrosis is a major contributor to chronic heart failure, which is a major cause of death worldwide. Our research has identified a promising therapeutic target in the development of new treatments for patients with chronic heart failure."

Professor Jeremy Pearson, Associate Medical Director at the British Heart Foundation said: "Scarring of the heart muscle has a major impact on the heart's ability to pump effectively. This can lead to heart failure and there is no effective treatment at the moment. By finding a new way to limit scarring in the hearts of mice, this research has unlocked the route to a potential treatment for this life-threatening problem."

http://bit.ly/2hh8QJ9

No magic wand required: Scientists propose way to turn any cell into any other cell type

By harnessing massive amounts of data on activity within and between snippets of DNA, researchers say it could be possible to reprogram both healthy and diseased cells

ANN ARBOR, Mich. -- In fairy tales, all it takes to transform a frog into a prince, a servant into a princess or a mouse into a horse is the wave of a magic wand.

But in the real world, transforming one living thing into another isn't so easy. Only in recent years have scientists discovered how to do it, with tiny individual living cells.

In fact, the team that figured it out won the Nobel Prize, for discovering how to take an ordinary human skin cell and transform it into a stem cell -- the same kind of cell found in embryos. With painstaking effort, such cells can grow up to become any other kind of cell in the body.

And in the last decade, that time-consuming transformation technique has opened the door for discoveries about many diseases, from birth defects to cancer. But what if scientists could cut out a step, and go straight from skin cell to any other kind of cell?

A new paper in the Proceedings of the National Academy of Sciences lays out a way to do it - and avoid all the intermediate steps involved in the other technique, which produces induced pluripotent stem cells.

In the paper, they lay out a way to harnessing the wealth of data now available about DNA activity, and reprogram cells directly. The formula also provides a blueprint for determining the optimal combination of factors and when they should be added to accomplish this reprogramming. Using this formula, the authors were able deduce the factors that the Nobel-winning team discovered, a process that required many years of trial and error.

The concept, developed by a team of University of Michigan scientists together with colleagues from the University of Maryland and Harvard University, combines biological information on genome structure and gene expression with a fair bit of math, using an approach called data-guided control. The paper's authors include world experts on control theory Roger Brockett, Ph.D. of Harvard and U-M mathematics department chair Anthony Bloch, Ph.D.

Though the paper spells out an algorithm for transforming cells -- and successfully predicts factors that are already known to reprogram cells -- it does not directly test the formula in the laboratory. The authors have plans to further test their method, and hope that it can be tried by scientists at Michigan and around the world.

If it bears fruit, they predict it could have applications including regenerating diseased or lost tissue, and fighting cancer.

"Cells in our body naturally specialize," says Indika Rajapakse, Ph.D., the U-M bioinformatics and mathematics researcher who is senior author of the new paper. "What we propose could provide a shortcut to doing the same, to help any cell become a targeted cell type."

Rajapakse notes that the idea of direct reprogramming is not new. In the late 1980s, a team led by the late scientist Harold Weintraub turned skin cells directly into muscle cells by bathing the cells in a type of molecule that encouraged certain genes in the cells' DNA to be "read". Rajapakse trained with Weintraub's colleague Mark Groudine, Ph.D. at Fred Hutchinson Cancer Research Center.

The new model builds on that idea, by also harnessing the power of these molecules, called transcription factors or TFs.

But instead of bathing the whole cell culture in one TF, the scientists aim to target cells with specific TFs at specific crucial times in their lifespan. They lay out a mathematical control model for harnessing all the information that can now be learned about cells at the molecular level, and combining it to map out the timing and sequence for injecting TFs to get the desired cell type.

"We have so much data now from RNA and transcription factor activity, and from Hi-C data of chromosome configuration that tells us how often two pieces of chromatin are near one another, that we

believe we can go from the cell's initial configuration to the desired configuration," says Rajapakse.

The Hi-C technique lets scientists track the location of, and contact between, portions of the DNA/protein complex called the chromatin. So even if two genes sit far apart on a long strand of DNA, they may come in close contact with one another when those looping, folding strands end up next to one another. If one of those genes gets "read", it may produce a transcription factor that then sets in motion the "reading" of the other gene, and the production of a certain protein that plays a key role in transforming the cell in some way.

The amount of data that would come out of analyzing these "topologically associating domains" in just one type of cell is huge. But modern bioinformatics techniques make it easier to make sense of it all.

The first author of the paper is Scott Ronquist, a Ph.D. student who began working with Rajapakse in the Computational Medicine and Bioinformatics department as an undergraduate at U-M. He and former postdoctoral fellow Geoff Patterson, Ph.D., led the effort to use gene expression and TAD data generated in the Rajapakse lab and publicly available gene expression and TF data to test their model. They were able to see patterns in the data that reflected the pace of normal cell differentiation.

Now, they're working on testing the model proactively, in the laboratory of Max Wicha, M.D., the Forbes Professor of Oncology at Michigan Medicine, U-M's academic medical center, and former director of the U-M Comprehensive Cancer Center.

"This algorithm provides a blueprint that has important implications for cancer, in that we think cancer stem cells may arise from normal stem cells via similar reprogramming pathways," says Wicha, who is a co-author on the PNAS paper. "This work also has important implications for regenerative medicine and tissue engineering, since it provides a blueprint for generating any desired cell type It also demonstrates the beauty of combining mathematics and biology to unravel the mysteries of nature."

NOTE: The University has filed for a patent on the algorithm. The work was funded by the Defense Advanced Research Projects Agency, or DARPA.

http://bit.ly/2qHF3IM

Infographic: how cannabis works

Researchers have unearthed receptors in the brain and body, that respond to THC, the psychoactive ingredient of cannabis.

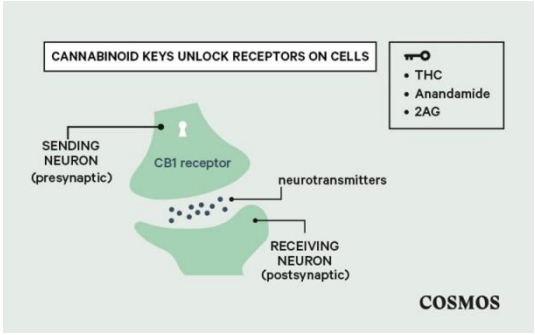
Any medicine, touted for everything from autism to asthma, sounds like snake oil. But there is biological plausibility for these claims.

Researchers have unearthed receptors in the brain and body, that respond to THC, the psychoactive ingredient of cannabis. Pot works because it is mimicking what home-made molecules – the endocannabinoids like anandamide and 2 arachidonoyl glycerol – do.

CB1 receptors, which are mostly in the brain, regulate areas involved with mood, memory appetite and movement.

CB2 receptors are mostly found in muscle, bones, the liver and the immune system. When activated, they tend to have beneficial effects, like toning down inflammation.

So we seem to be have been gifted with an endocannabinoid system that blisses out our brain and ratchets down inflammation in the body. Why? “It seems to have a protective role,” suggests Roger Pertwee, a pharmacologist at the University of Aberdeen who has pioneered the study of the endocannabinoid system.



Cannabis keys unlock receptors on cells **Cosmos Magazine**

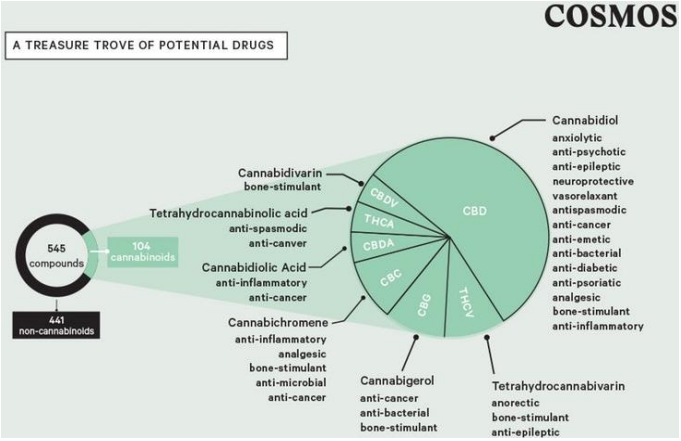
Cannabinoid keys unlock receptors on cells

Until 1964, we had no idea how cannabis works. In that year, Raphael Mechoulam at Hebrew University in Jerusalem and his team, isolated the psychoactive ingredient THC. THC works like a key that unlocks “receptors” on the surface of different types of cells. By unlocking

receptors on different brain neurons THC creates sensations ranging from paranoia to euphoria and the munchies. The natural keys for these locks are the endocannabinoids “anandamide” discovered by Mechoulam’s group in 1992 and 2- arachidonoyl glycerol, discovered by the same group in 1995.

A treasure trove of potential drugs

Cannabis contains more than 500 chemicals. There are 104 cannabinoids unique to the plant as well as flavonoids, terpenes and fatty acids. Research is focused on the non-psychoactive cannabinoids shown. At present, it’s not clear how these non-psychoactive compounds act.

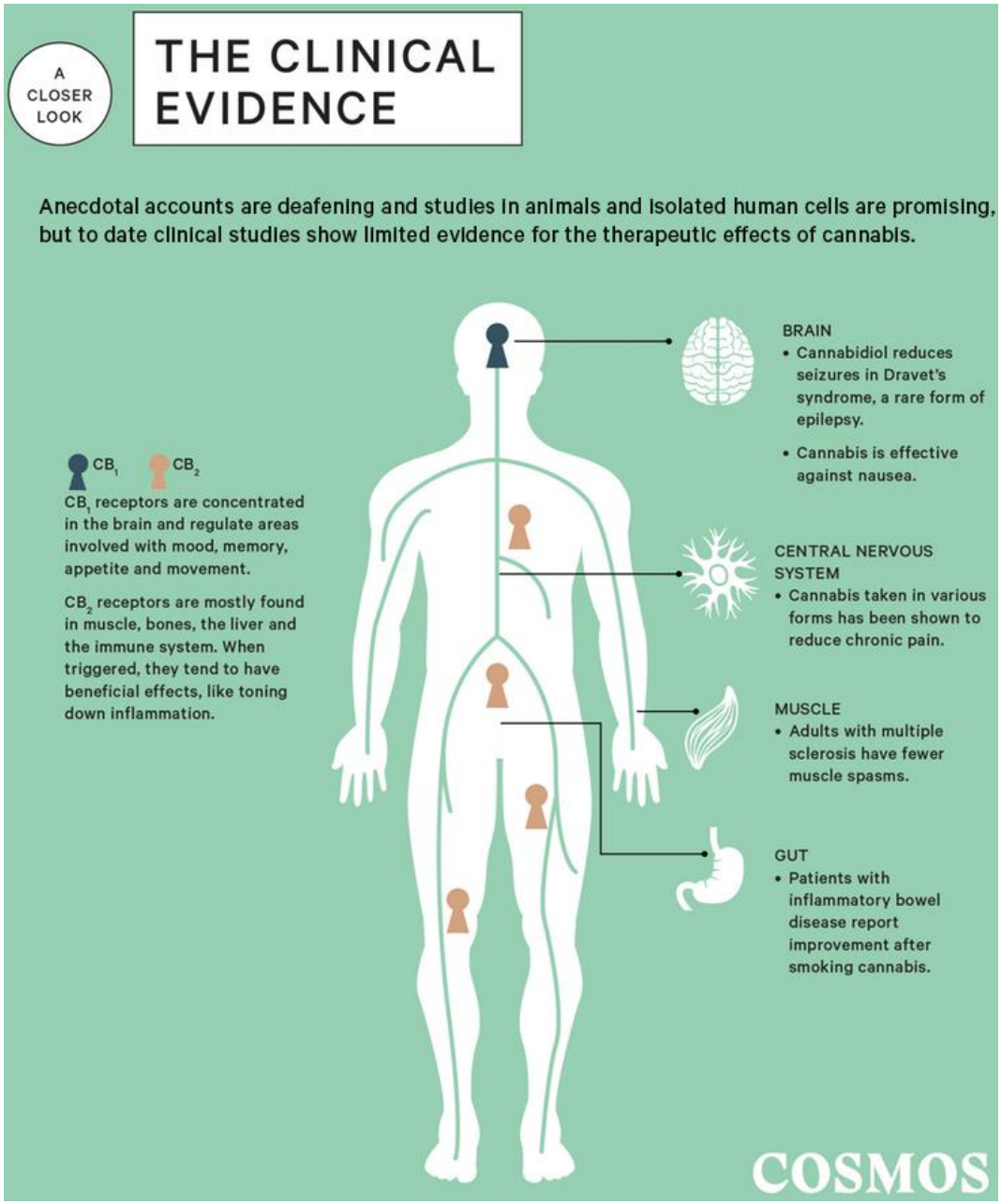


The treasure trove of potential drugs from cannabis **Cosmos Magazine, Adapted from izzo et al. 2009**

They don’t unlock the same receptor as THC, nor do they seem to have their own receptors. There is some evidence that cannabidiol, modifies the THC receptor. Most of the evidence for the effects of these compounds comes from animal studies or from cells growing in a dish.

A closer look at the clinical evidence of medical cannabis

When it comes to the actual effects of cannabis compounds on human disease, so far there is very little solid evidence. Modest evidence now exists for the usefulness of cannabis in a rare form of epilepsy, nausea, chronic pain, Crohn’ disease and the muscle spasms of multiple sclerosis.



A closer look at the clinical evidence of medical cannabis. **Cosmos Magazine**

http://bbc.in/2yXyzjo

Typhoid vaccine set to have 'huge impact'

A new vaccine that could prevent up to nine-in-10 cases of typhoid fever has been recommended by the World Health Organization.

By James Gallagher Health and science reporter, BBC News website

Experts say it could have a "huge impact" on the 22 million cases, and 220,000 deaths, from typhoid each year. Crucially it works in children, who are at high-risk of the infection, unlike other typhoid vaccines. It is hoped the vaccine could eventually help countries eliminate typhoid.

Typhoid fever is caused by Salmonella Typhi bacteria and patients have:

prolonged fever	***headache***
nausea	***loss of appetite***
constipation	***in one-in-100 cases it causes fatal complications***

The bacteria are highly contagious and spread through contaminated food or water.

The infection is most common in countries with poor sanitation and a lack of clean water, particularly in south Asia and sub-Saharan Africa.

Two typhoid vaccines already approved to help reduce the number of cases, but none are licensed for children under the age of two.

The decision to recommend the new conjugate typhoid vaccine was made by the WHO's Strategic Advisory Group of Experts on Immunization (Sage). Prof Alejandro Cravioto, the chairman of Sage, said: "For the first time I think we do have a very effective vaccine."

Sage recommended the vaccine should be given to children aged six-months old and said catch-up campaigns focusing on children up to 15 years old should also take place.

Prof Cravioto said the vaccine was vital as the world was "reaching the limit" of current treatments due to the "crazy amount" of antibiotic resistance the typhoid bacterium had acquired.

'A valuable weapon'

Data from a clinical trial of the vaccine, carried out by the University of Oxford, was published just last month in the Lancet medical journal.

The "challenge study" gave the vaccine to 112 people and then deliberately infected (challenged) them with typhoid-causing bacteria.

Such an approach gives a clear picture of a vaccine's effectiveness without having to immunise thousands of people. It showed the vaccine was up to 87% effective. Prof Andrew Pollard, who ran those trials, told the BBC News website: "It could have a huge impact.

"If it can be rolled out in all communities, it must be possible to prevent the majority of cases and if it also interrupts transmission, which hasn't been shown yet, then it could be eliminated from those regions."

Gavi, the global body that pays for vaccines in 68 low and middle-income countries, is now considering whether to add the typhoid vaccine.

Dr Seth Berkley, the chief executive of Gavi, said: "Not only could this vaccine save lives, it could also prove to be a valuable weapon in the fight against antimicrobial resistance."

Before antibiotics, typhoid killed one-in-five people infected. Now there is growing levels of typhoid that is resistant to drugs.

http://bit.ly/2xuKqKM

Artificial intelligence tells nightmare-inducing tales of terror

I remember his face in a look of horror, and it was agony and malice. I was trapped. I was trapped in this hospital bed.

By Timothy Revell

This isn’t the start of the latest Stephen King novel, it’s in fact the start of a horror story told by artificial intelligence.

Shelley AI is currently working on a different short scary tale once an hour leading up to Halloween. It’s powered by deep learning algorithms that have been trained on stories collected from the subreddit /r/nosleep where people share their own original eerie works.

The AI was created by researchers at the Massachusetts Institute of Technology Media Lab.

The AI works best when given a bit of inspiration so anyone can join in the story writing process to make it a joint effort. Collaborators simply add their own blood-curdling sentences to the compositions so far on Twitter and Shelley then continues from where they finish.

Some of the best sinister snippets so far include:

I heard my mother's voice scream from the darkness. "God dad, please don't hurt me again. Please don't leave me alone. Please. I don't want to be here anymore." I turned to my left, and for a split second I could see it was her. After all that time, all I could make out were those black eyes above me.

My heart is beating so fast it is a bit shorter than my breathing. I think I'm being stalked. I don't know what happened to me, or why I was scared of it, but I need to know why. I need to know what happened.

And my personal favourite:

She fell to the floor from her cries and muttered a soft 'Come to meeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeeee eeeeeeeeeeee'.

http://bit.ly/2gUg6h8

Some dead bodies donated to research in US end up in warehouses of horrors

Despite egregious ethics, law enforcement can do nothing. Families left aghast.

Beth Mole - 10/25/2017, 1:55 AM

Dead human bodies are critical to medical training, physicians and researchers say. And thousands of Americans are happy to donate their meat suits for the greater good after they're gone. But in the US, a body's trip from a morgue to a medical school or lab can be gruesome, shady, and expensive.

Some don't make it at all. Instead, bits and pieces of donated loved ones—sometimes disassembled with chainsaws—end up decomposing in parking lots, forgotten in unplugged freezers, and tossed unceremoniously into incinerators.

And law enforcement can do nothing—there are few to no laws that regulate the grim industry of human body brokering. Grieving families, who are often misled and in the dark about the fate of their loved ones, can be left horrified.

That's all according to a new investigative report by Reuters, which tracked the practices and pricing of dozens of such brokers across the country. They found that the lucrative business includes a bloody splattering of practices—some ethical, lots not—that can bring in millions to even the most shoddy and small brokers.

Basically, what happens is that these companies approach dying people or families of the recently deceased, usually through a funeral home connection. The companies give emotional sales pitches to convince people to donate—for free—a human body and get a deal on funeral costs. The companies then take the bodies to some type of unregulated facility where they're kept whole or dismembered and sold—for profit—to medical training facilities, research labs, and other buyers.

Whole bodies can go for anywhere from $3,000 to $5,000, and sometimes as high as $10,000. But it's often more profitable to sell chunks. A torso with legs can go for $3,750, a head may go for $500, and a spine may set a buyer back $300. Companies claim that the prices simply cover their operating fees, but documents obtained by Reuters show that many companies see bodies and parts as commodities.

Rolling heads?

There are few laws governing how this whole process should play out from beginning to end. Bodies can be butchered with proper surgical tools or with chainsaws. They can be responsibly scanned for diseases and surgical implants or not. They can be properly stored in freezers or left out to decompose. If parts go unsold, they can be carelessly incinerated. And family members may not know about any of this.

Last year, Reuters reported that a body broker in Arizona sold 20 bodies to the US Army, which used them for blast experiments. The

family members had no idea, and some thought their loved ones' remains were being used to help study diseases, like Alzheimer's. Some had specifically noted that they didn't want the bodies to be used in military trials. They learned of the blast experiments from the Reuters report.

"The current state of affairs is a free-for-all," Angela McArthur told Reuters. McArthur directs the body-donation program at the University of Minnesota Medical School and formerly chaired Minnesota's anatomical donation commission. "We are seeing similar problems to what we saw with grave-robbers centuries ago… I don't know if I can state this strongly enough: what they are doing is profiting from the sale of humans."

Todd Olson, an anatomy and structural biology professor at Yeshiva University's Albert Einstein College of Medicine, echoed the statement. He emphasized that there are no consistent state or federal laws governing how these brokers do business. "Nobody is accounting for anything, nobody is watching. We regulate heads of lettuce in this country more than we regulate heads of bodies."

The daughter of a New Mexico man who donated his body to Albuquerque broker Bio Care said she and her father were told the body would be used to help train doctors and that she would receive some cremated remains. Instead, she received a jar of sand—not ashes—and learned that Bio Care was in the body part-selling business.

Her father's head was among a pile of parts in a medical incinerator recovered later by authorities. The parts appeared to have been cut by a "coarse cutting instrument, such as a chainsaw," a police detective wrote in an affidavit.

Authorities charged Bio Care owner Paul Montano with fraud but later dropped the case, saying that no laws were broken.

"It's not OK," said Kari Brandenburg, a former district attorney in Albuquerque. "But it doesn't make it a crime. There's no criminal law that says this is wrong."

http://bit.ly/2lpzbcv

New Gene-Editing "Pencil" Erases Disease-Causing Errors

This tool could, in theory, fix genetic mistakes that lead to about 15,000 illnesses

By Karen Weintraub on October 25, 2017

There are more than 50,000 known human genetic maladies that have, in most cases, few good treatments and no cure. Now researchers at the Broad Institute of Harvard and MIT have developed a new tool that would theoretically make it possible to correct the genetic errors behind about 15,000 of these illnesses—including sickle-cell disease, cystic fibrosis and several forms of congenital deafness and blindness.

Standard gene-editing tools, such as the well-known CRISPR–Cas9 system, function like scissors; they can cut an offending gene from a strand of DNA. This could be useful against diseases such as Huntington's, which is caused by duplications of genetic material.

The new tool, called ABE (adenine base editors), is more like an editing pencil, according to lead researcher David Liu. It lets scientists precisely change individual pairs of bases—the "letters" that form the "sentences" of the vast human genome—and thus might help address diseases like sickle cell that can be treated with a single letter change. Liu emphasizes that one tool is not better than the other; rather they can be used to address different types of problems.

But before ABE can be tried in human patients, Liu says, doctors would need to determine when to intervene in the course of a genetic disease. They would also have to figure out how to best deliver the gene editor to the relevant cells—and to prove the approach is safe and effective enough to make a difference for the patient.

Genes are made up of DNA—two long, parallel strands of molecules called nucleotides that are linked by pairs of chemical bases. The base A (adenine) always pairs with T (thymine); and G (guanine) joins with C (cytosine). But when the genetic machinery makes mistakes and

puts a pair in the wrong place, it sometimes leads to disease. The new tool targets genetic errors in which an A–T base pair should be a G–C. Liu is a professor of chemistry at Harvard University and a vice chair of the faculty at the Broad Institute. Along with his students and postdoctoral researchers he had previously developed base editors that convert C–G base pairs into T–A pairs. (The order is important, so a G–C mistake is not the same as a C–G one.) Liu, who is also an investigator with the Howard Hughes Medical Institute, said Tuesday in a news conference that he and others have been working on additional tools, which could correct other types of "spelling mistakes" in DNA. This led them to ABE.

The new ABE technique uses an enzyme Liu and his colleagues developed. It rearranges the atoms in A so they form a base that resembles G in a DNA strand. The ABE system also nicks the mated DNA strand that contains the T. The cell's repair mechanisms then turn on to fix the tear. In doing so, the cell replaces the T with a C, correcting the other half of the base pair. The net result is the troublesome A–T base pair is converted into a beneficial G–C pair.

Using ABE in a lab dish, Liu and his colleagues were able to precisely edit genes that cause a hereditary form of hemochromatosis—a disease that leads the body to store too much iron, causing pain, fatigue, weakness and, if untreated, liver and heart failure. They also used ABE to install a different genetic mutation that compensates for the DNA defect that causes sickle-cell disease.

The ABE gene-editing process is efficient, effectively editing the relevant spot in the genome an average of 53 percent of the time across 17 tested sites, Liu said. It caused undesired effects less than 0.1 percent of the time, he added. That success rate is comparable with what CRISPR can do when it is cutting genes.

Dirk Hockemeyer, an assistant professor at the University of California, Berkeley, who was not involved in the Broad research, said he is impressed in the work and the tool the team developed. But it is still a long way from helping patients. "In clinical applications the key question is always delivery, delivery, delivery: How do I get the editing agent to the position in the cell that I want to repair?" he says. But "if it cures a single disease, we should all be happy."

http://bit.ly/2qLqvZ2

Individual with complete spinal cord injury regains voluntary motor function

Extended activity-based training with epidural stimulation resulted in ability to stand and move without stimulation

LOUISVILLE, Ky. - A research participant at the University of Louisville with a complete spinal cord injury, who had lost motor function below the level of the injury, has regained the ability to move his legs voluntarily and stand six years after his injury.

A study published today in Scientific Reports describes the recovery of motor function in a research participant who previously had received long-term activity-based training along with spinal cord epidural stimulation (scES).

In the article, senior author Susan Harkema, Ph.D., professor and associate director of the Kentucky Spinal Cord Injury Research Center (KSCIRC) at the University of Louisville, and her colleagues report that over the course of 34.5 months following the original training, the participant recovered substantial voluntary lower-limb motor control and the ability to stand independently without the use of scES.

"Activity-dependent plasticity can re-establish voluntary control of movement and standing after complete paralysis in humans even years after injury," Harkema said. "This should open up new opportunities for recovery-based rehabilitation as an agent for recovery, not just learning how to function with compensatory strategies, even for those with the most severe injuries."

Previous research at KSCIRC involving four participants with chronic clinically motor-complete spinal cord injury found that activity-based training with the use of scES - electrical signals delivered to motor neurons in the spine by an implanted device - allowed the participants

to stand and to perform relatively fine voluntary lower limb movements when the scES device was activated.

Andrew Meas was one of the four participants in that study.

The original training protocol included daily one-hour activity-based training sessions with the aid of epidural stimulation. During these sessions, the participant trained on standing activity for several months, followed by several months of training on stepping.

After completing a nine-month training program in the lab, Meas continued activity-based stand training at home. After a year of independent training, he returned to the lab to train for three months in a revised activity-based training schedule. The revised training called for two daily one-hour training sessions and included both stand and step training each day, all with the aid of epidural stimulation.

After that training, Meas was able to voluntarily extend his knees and his hip flexion was improved. In addition, using his upper body and minimal additional assistance to reach a standing position, he was able to remain in a standing position without assistance and even stand on one leg, without the use of epidural stimulation.

"We observed that in participants we have worked with so far, eight months of activity-based training with stimulation did not lead to any improvement without stimulation," said Enrico Rejc, Ph.D., assistant professor in the UofL Department of Neurological Surgery and the article's first author.

"This participant kept training at home and, after several months, he came back to the lab and we tried a different training protocol. After a couple of months of training with the new protocol, we surprisingly observed that he was able to stand without any stimulation - with two legs and with one leg - using only his hands for balance control."

The authors suggest that several mechanisms may be responsible for Meas's recovery of mobility, including the sprouting of axons from above the point of injury into areas below the lesion. Another possible explanation may be that the activity-based training with scES promoted remodelling of connections among neurons in the spinal cord.

In addition, they suggest that the participant's own effort at voluntary movement may have been a factor in the recovery. During the revised training, Meas was attentive and focused on the trained motor task, actively attempting to contribute to the motor output.

"The voluntary component of him trying constantly with spinal stimulation on and while performing motor tasks can lead to unexpected recovery," Rejc said.

"The human nervous system can recover from severe spinal cord injury even years after injury. In this case, he was implanted with the stimulator four years after his injury. We saw motor recovery two years later--so six years after injury," Rejc said. "It is commonly believed that one year from injury, you are classified as chronic and it's likely that you will not improve any more. This data is proof of principle that the human nervous system has much greater recovery capabilities than expected."

Funding for the research in Harkema's lab is supported by the Christopher & Dana Reeve Foundation, the Leona B. and Harry B. Helmsley Charitable Trust, Medtronic and the National Institutes of Health.

"We are enormously excited about this development in Dr. Harkema's work, as it not only validates the promise of effective treatments for spinal cord injury, but further demonstrates the spinal cord's ability to recover after severe trauma," said Peter Wilderotter, president & CEO of the Christopher & Dana Reeve Foundation.

"As we continue to support and fund Dr. Harkema's research, it is awe-inspiring to see another breakthrough on the path to cures for paralysis, and how much this particular treatment has improved quality of life and health for Drew."

For more information on epidural stimulation research, visit https://www.reevebigidea.org/

http://nyti.ms/2qLMWxb

C.D.C. Panel Recommends a New Shingles Vaccine

In an unusually close vote, an advisory panel to the Centers for Disease Control and Prevention on Wednesday recommended the use of a new vaccine to prevent shingles over an older one that was considered less effective.

By SHEILA KAPLAN OCT. 25, 2017

WASHINGTON - The decision was made just days after the Food and Drug Administration announced approval of the new vaccine, called Shingrix and manufactured by GlaxoSmithKline, for adults ages 50 and older. The panel's recommendation gives preference to the new vaccine over Merck's Zostavax, which has been the only shingles vaccine on the market for over a decade and was recommended for people ages 60 and older.

The Advisory Committee on Immunization Practices also recommended that adults who have received the older vaccine get the new one. Even with the committee vote, this recommendation still awaits formal endorsement by the head of the C.D.C., which usually takes a couple of months. Insurance companies must also agree to cover the cost of the vaccine, which GSK estimates to be $280 for two doses.

According to the C.D.C., almost one of every three people in the United States will contract shingles, a viral infection that can result in a painful rash and lasting nerve damage.

The disease, also known as herpes zoster, can range in severity from barely noticeable to debilitating. It is caused by the varicella-zoster virus, which also causes chickenpox. Once a person has had chickenpox, the virus lies inactive in nerve tissue. Years later, it may reactivate as shingles. The C.D.C. estimates that about one million cases are diagnosed in the United States each year.

"This is what we've been waiting for," said Dr. Anne Louise Oaklander, an associate professor of neurology at Harvard Medical School and an expert in the disease. "Shingles is an unappreciated and common cause of severe problems throughout the nervous system."

Dr. Oaklander said that while rash symptoms lead some people to consider shingles as minor as a bad sunburn, the illness can cause strokes, encephalitis, spinal cord damage and loss of vision.

A new shingles vaccine called Shingrix, manufactured by GlaxoSmithKline, was recommended by a C.D.C. panel on Wednesday. **GlaxoSmithKline, via Associated Press**

Given in one dose, Zostavax had shown a 51 percent reduction in shingles and a 67 percent reduction in nerve pain. Shingrix is given in two doses, and the company said clinical trials showed it to be about 98 percent effective for one year and about 85 percent over three years. By preventing shingles, the vaccine also drastically reduces the overall incidence of severe nerve pain, a lasting complication for about one in three people who get shingles. GlaxoSmithKline said it tested the vaccine in more than 38,000 people.

"We believe Shingrix will provide confidence in the protection one can expect from a shingles vaccine," said Luc Debruyne, the company's president of global vaccines.

The recommendation of the advisory committee will be considered an endorsement of Shingrix over Zostavax, although the closeness of the committee vote, 8 to 7, may mitigate the market loss for Merck.

Dr. Kathleen Dooling, a medical officer in the C.D.C.'s division of viral disease, said she expected the agency's final recommendation to be issued early next year.

"The Shingrix vaccine has the potential to prevent tens of thousands of cases of shingles and its complications," Dr. Dooling said.

She cautioned, however, that more people had adverse reactions to Shingrix than to Zostavax, including fever and muscle aches.

“Patients and health care providers should be aware that this vaccine is very effective, but it also causes more reactions than they may be used to with other adult vaccines,” she said. “All indications are these are not dangerous to one’s health, but they may interfere with your daily activities for a few days.”

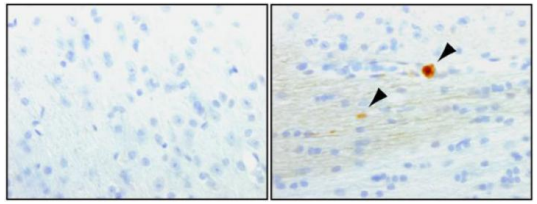
GlaxoSmithKline said its new vaccine would cost about $280 and would be available next month. Zostavax costs about $223.

http://bit.ly/2zOefOV

Scientists find a role for Parkinson's gene in the brain

NIH-funded mouse study suggests LRRK gene is needed for dopamine neuron health

A new study published in the journal Neuron sheds light on the normal function of LRRK2, the most common genetic cause for late-onset Parkinson's disease. The study was supported by the National Institute of Neurological Disorders and Stroke (NINDS), part of the National Institutes of Health.



In the region of the brain affected by Parkinson's disease, mice lacking LRRK (right) have an increase in cell death (brown; arrowheads) compared to normal mice (left). **Image courtesy of Shen lab**

For more than 10 years, scientists have known that mutations in the LRRK2 gene can lead to Parkinson's disease, yet both its role in the disease and its normal function in the brain remain unclear. In a study in mice, researchers have now found that LRRK is necessary for the survival of dopamine-containing neurons in the brain, the cells most affected by Parkinson's. Importantly, this finding could alter the design of treatments against the disease.

"Since its discovery, researchers have been trying to define LRRK2 function and how mutations may lead to Parkinson's disease," said Beth-Anne Sieber, Ph.D., program director at NINDS. "The findings in this paper emphasize the importance of understanding the normal role for genes associated with neurodegenerative disorders."

LRRK2 is found along with a closely related protein, LRRK1, in the brain. A mutation in LRRK2 alone can eventually produce Parkinson's disease symptoms and brain pathology in humans as they age. In mice, however, LRRK2 loss or mutation does not lead to the death of dopamine-producing neurons, possibly because LRRK1 plays a complementary or compensatory role during the relatively short, two-year mouse lifespan.

"Parkinson's-linked mutations such as LRRK2 have subtle effects that do not produce symptoms until late in life. Understanding the normal function of these types of genes will help us figure out what has gone wrong to cause disease," said Jie Shen, Ph.D., director of the NINDS Morris K. Udall Center of Excellence for Parkinson's Disease at Brigham and Women's Hospital and senior author of this study.

To better understand the roles of these related proteins in brain function using animal models, Shen and her colleagues created mice lacking both LRRK1 and LRRK2. They observed a loss of dopamine-containing neurons in areas of the brain consistent with PD beginning around 15 months of age. When the researchers looked at the affected brain cells more closely, they saw the buildup of a protein called α-synuclein, a hallmark of Parkinson's, and defects in pathways that clear cellular "garbage." At the same time, more dopamine-containing neurons also began to show signs of apoptosis, the cells' "self-destruct" mechanism.

"Our findings show that LRRK is critical for the survival of the populations of neurons affected by Parkinson's disease," said Dr. Shen. While the deletion of both LRRK1 and LRRK2 did not affect overall brain size or cells in such areas of the brain as the cerebral cortex and cerebellum, the mice showed other significant effects such as a decrease in body weight and a lifespan of only 15 to 16 months. Thus, the scientists were unable to study other Parkinson's-related effects

such as changes in behavior and movement nor were they able to conduct a long-term analysis of how LRRK's absence affects the brain. Interestingly, the most common disease-linked mutation in LRRK2 is thought to make the protein more active. As a result, most efforts to develop a treatment against that mutation have focused on inhibiting LRRK2 activity.

"The fact that the absence of LRRK leads to the death of dopamine-containing neurons suggests that the use of inhibitory drugs as a treatment for Parkinson's disease might not be the best approach," said Dr. Shen.

Dr. Shen and her colleagues are now developing mice that have LRRK1 and 2 removed only in the dopamine-containing neurons of the brain. This specific deletion will allow the researchers to study longer-term and behavioral changes while avoiding the other consequences that lead to a shortened lifespan.

This study was supported by the NINDS (NS071251, NS094733)

Article: Giaime et al. Age-dependent dopaminergic neurodegeneration and impairment of the autophagy-lysosomal pathway in LRRK-deficient mice. Neuron. October 19, 2017 DOI: 10.1016/j.neuron.2017.09.036

http://bit.ly/2qKY4Ks

Dogs may protect against childhood eczema and asthma

Eczema protection reduces as children grow

BOSTON, MA - "Good dog!" Two studies being presented at the American College of Allergy, Asthma and Immunology (ACAAI) Annual Scientific Meeting show there may be even more reason to love your dog. The first study shows babies born in a home with a dog during pregnancy receive protection from allergic eczema, though the protective effect goes down by age 10. A second study shows dogs may provide a protective effect against asthma, even in children allergic to dogs.

"Although eczema is commonly found in infants, many people don't know there is a progression from eczema to food allergies to nasal allergies and asthma," says allergist Gagandeep Cheema, MD, ACAAI member and lead author. "We wanted to know if there was a protective effect in having a dog that slowed down that progress."

The study examined mother-child pairs exposed to a dog. "Exposure" was defined as keeping one or more dogs indoors for at least one hour daily. "We found a mother's exposure to dogs before the birth of a child is significantly associated with lower risk of eczema by age 2 years, but this protective effect goes down at age 10," says allergist Edward M. Zoratti, MD, ACAAI member and a study co-author.

In the second study, researchers examined the effects of two different types of dog exposure on children with asthma in Baltimore. The first type was the protein, or allergen, that affects children who are allergic to dogs. The second type were elements, such as bacteria, that a dog might carry. The researchers concluded that exposure to the elements that dogs carry may have a protective effect against asthma symptoms. But exposure to the allergen may result in more asthma symptoms among urban children with dog allergy.

"Among urban children with asthma who were allergic to dogs, spending time with a dog might be associated with two different effects," says Po-Yang Tsou, MD, MPH, lead author. "There seems to be a protective effect on asthma of non-allergen dog-associated exposures, and a harmful effect of allergen exposure." The researchers believe that a child's contact with factors other than dog allergen, such as bacteria or other unknown factors, may provide the protective effect. "However, dog allergen exposure remains a major concern for kids who are allergic to dogs," says Dr. Tsou.

People with dog allergy should work with their allergist to reduce exposure. ACAAI has additional tips for those with dog allergy who keep a dog in the home:

Keep your dog out of your bedroom and restrict it to only a few rooms. But know that keeping the dog in only one room will not limit the allergens to that room.

After you pet or hug your dog, wash your hands with soap and water.

High-efficiency particulate air (HEPA) cleaners that run continuously in a bedroom or living room can reduce allergen levels over time. Regular

use of a high-efficiency vacuum cleaner or a central vacuum can also reduce allergen levels.

Giving your dog a bath at least once a week can reduce airborne dog allergen.

Abstract Title: *Effect of Prenatal Dog Exposure on Eczema Development in Early and Late Childhood. Author: Gagandeep Cheema, MD*

Abstract Title: *The Effect of Animal Exposures on Asthma Morbidity Independent of Allergen Among Inner-city Asthmatic Children. Author: Po-Yang Tsou, MD, MPH*

http://bit.ly/2xwPnKu

The Unforgiving Math That Stops Epidemics

If you didn't get a flu shot, you are endangering more than just your own health. Calculations of herd immunity against common diseases don't make exceptions.

Tara C. Smith

As the annual flu season approaches, medical professionals are again encouraging people to get flu shots. Perhaps you are among those who rationalize skipping the shot on the grounds that "I never get the flu" or "if I get sick, I get sick" or "I'm healthy, so I'll get over it." What you might not realize is that these vaccination campaigns for flu and other diseases are about much more than your health. They're about achieving a collective resistance to disease that goes beyond individual well-being — and that is governed by mathematical principles unforgiving of unwise individual choices.

When talking about vaccination and disease control, health authorities often invoke "herd immunity." This term refers to the level of immunity in a population that's needed to prevent an outbreak from happening. Low levels of herd immunity are often associated with epidemics, such as the measles outbreak in 2014-2015 that was traced to exposures at Disneyland in California. A study investigating cases from that outbreak demonstrated that measles vaccination rates in the exposed population may have been as low as 50 percent. This number was far below the threshold needed for herd immunity to measles, and it put the population at risk of disease.

The necessary level of immunity in the population isn't the same for every disease. For measles, a very high level of immunity needs to be maintained to prevent its transmission because the measles virus is possibly the most contagious known organism. If people infected with measles enter a population with no existing immunity to it, they will on average each infect 12 to 18 others. Each of those infections will in turn cause 12 to 18 more, and so on until the number of individuals who are susceptible to the virus but haven't caught it yet is down to almost zero. The number of people infected by each contagious individual is known as the "basic reproduction number" of a particular microbe (abbreviated R_0), and it varies widely among germs. The calculated R_0 of the West African Ebola outbreak was found to be around 2 in a 2014 publication, similar to the R_0 computed for the 1918 influenza pandemic based on historical data.

If the Ebola virus's R_0 sounds surprisingly low to you, that's probably because you have been misled by the often hysterical reporting about the disease. The reality is that the virus is highly infectious only in the late stages of the disease, when people are extremely ill with it. The ones most likely to be infected by an Ebola patient are caregivers, doctors, nurses and burial workers — because they are the ones most likely to be present when the patients are "hottest" and most likely to transmit the disease. The scenario of an infectious Ebola patient boarding an aircraft and passing on the disease to other passengers is extremely unlikely because an infectious patient would be too sick to fly. In fact, we know of cases of travelers who were incubating Ebola virus while flying, and they produced no secondary cases during those flights.

Note that the R_0 isn't related to how severe an infection is, but to how efficiently it spreads. Ebola killed about 40 percent of those infected in West Africa, while the 1918 influenza epidemic had a case-fatality rate of about 2.5 percent. In contrast, polio and smallpox historically spread to about 5 to 7 people each, which puts them in the same range

as the modern-day HIV virus and pertussis (the bacterium that causes whooping cough).

Determining the R_0 of a particular microbe is a matter of more than academic interest. If you know how many secondary cases to expect from each infected person, you can figure out the level of herd immunity needed in the population to keep the microbe from spreading. This is calculated by taking the reciprocal of R_0 and subtracting it from 1. For measles, with an R_0 of 12 to 18, you need somewhere between 92 percent (1 – 1/12) and 95 percent (1 – 1/18) of the population to have effective immunity to keep the virus from spreading. For flu, it's much lower — only around 50 percent. And yet we rarely attain even that level of immunity with vaccination.

Once we understand the concept of R_0, so much about patterns of infectious disease makes sense. It explains, for example, why there are childhood diseases — infections that people usually encounter when young, and against which they often acquire lifelong immunity after the infections resolve. These infections include measles, mumps, rubella and (prior to its eradication) smallpox — all of which periodically swept through urban populations in the centuries prior to vaccination, usually affecting children.

Do these viruses have some unusual affinity for children? Before vaccination, did they just go away after each outbreak and only return to cities at approximately five- to 10-year intervals? Not usually. After a large outbreak, viruses linger in the population, but the level of herd immunity is high because most susceptible individuals have been infected and (if they survived) developed immunity. Consequently, the viruses spread slowly: In practice, their R_0 is just slightly above 1. This is known as the "effective reproduction number" — the rate at which the microbe is actually transmitted in a population that includes both susceptible and non-susceptible individuals (in other words, a population where some immunity already exists). Meanwhile, new susceptible children are born into the population. Within a few years, the population of young children who have never been exposed to the disease dilutes the herd immunity in the population to a level below what's needed to keep outbreaks from occurring. The virus can then spread more rapidly, resulting in another epidemic.

An understanding of the basic reproduction number also explains why diseases spread so rapidly in new populations: Because those hosts lack any immunity to the infection, the microbe can achieve its maximum R_0. This is why diseases from invading Europeans spread so rapidly and widely among indigenous populations in the Americas and Hawaii during their first encounters. Having never been exposed to these microbes before, the non-European populations had no immunity to slow their spread.

Once we understand the concept of R_0, so much about patterns of infectious disease makes sense. It explains, for example, why there are childhood diseases.

If we further understand what constellation of factors contributes to an infection's R_0, we can begin to develop interventions to interrupt the transmission. One aspect of the R_0 is the average number and frequency of contacts that an infected individual has with others susceptible to the infection. Outbreaks happen more frequently in large urban areas because individuals living in crowded cities have more opportunities to spread the infection: They are simply in contact with more people and have a higher likelihood of encountering someone who lacks immunity. To break this chain of transmission during an epidemic, health authorities can use interventions such as isolation (keeping infected individuals away from others) or even quarantine (keeping individuals who have been exposed to infectious individuals — but are not yet sick themselves — away from others).

Other factors that can affect the R_0 involve both the host and the microbe. When an infected person has contact with someone who is susceptible, what is the likelihood that the microbe will be transmitted? Frequently, hosts can reduce the probability of transmission through their behaviors: by covering coughs or sneezes for diseases transmitted through the air, by washing their

contaminated hands frequently, and by using condoms to contain the spread of sexually transmitted diseases.

These behavioral changes are important, but we know they're far from perfect and not particularly efficient in the overall scheme of things. Take hand-washing, for example. We've known of its importance in preventing the spread of disease for 150 years. Yet studies have shown that hand-washing compliance even by health care professionals is astoundingly low — less than half of doctors and nurses wash their hands when they're supposed to while caring for patients. It's exceedingly difficult to get people to change their behavior, which is why public health campaigns built around convincing people to behave differently can sometimes be less effective than vaccination campaigns.

How long a person can actively spread the infection is another factor in the R_0. Most infections can be transmitted for only a few days or weeks. Adults with influenza can spread the virus for about a week, for example. Some microbes can linger in the body and be transmitted for months or years. HIV is most infectious in the early stages when concentrations of the virus in the blood are very high, but even after those levels subside, the virus can be transmitted to new partners for many years. Interventions such as drug treatments can decrease the transmissibility of some of these organisms.

The microbes' properties are also important. While hosts can purposely protect themselves, microbes don't choose their traits. But over time, evolution can shape them in a manner that increases their chances of transmission, such as by enabling measles to linger longer in the air and allowing smallpox to survive longer in the environment.

By bringing together all these variables (size and dynamics of the host population, levels of immunity in the population, presence of interventions, microbial properties, and more), we can map and predict the spread of infections in a population using mathematical models. Sometimes these models can overestimate the spread of infection, as was the case with the models for the Ebola outbreak in 2014. One model predicted up to 1.4 million cases of Ebola by January 2015; in reality, the outbreak ended in 2016 with only 28,616 cases. On the other hand, models used to predict the transmission of cholera during an outbreak in Yemen have been more accurate.

The difference between the two? By the time the Ebola model was published, interventions to help control the outbreak were already under way. Campaigns had begun to raise awareness of how the virus was transmitted, and international aid had arrived, bringing in money, personnel and supplies to contain the epidemic. These interventions decreased the Ebola virus R_0 primarily by isolating the infected and instituting safe burial practices, which reduced the number of susceptible contacts each case had. Shipments of gowns, gloves and soap that health care workers could use to protect themselves while treating patients reduced the chance that the virus would be transmitted. Eventually, those changes meant that the effective R_0 fell below 1 — and the epidemic ended. (Unfortunately, comparable levels of aid and interventions to stop cholera in Yemen have not been forthcoming.)

Catch-up vaccinations and the use of isolation and quarantine also likely helped to end the Disneyland measles epidemic, as well as a slightly earlier measles epidemic in Ohio. Knowing the factors that contribute to these outbreaks can aid us in stopping epidemics in their early stages. But to prevent them from happening in the first place, a population with a high level of immunity is, mathematically, our best bet for keeping disease at bay.

http://bit.ly/2zZ5AtV

Peat bogs defy the laws of biodiversity

Peat bogs play an essential role on Earth. Covering just 3% of surface, they store the equivalent of 67% of all the CO2 in the air.

Alexandre Buttler, head of EPFL's Ecological Systems Laboratory (ECOS), and Luca Bragazza, a scientist at ECOS, along with a team of European researchers including two former EPFL post-docs, analyzed 560 intact peat bog samples from 56 European countries to

study how peat bog ecosystems respond to different temperatures, precipitation levels and air pollution levels. They found that peat bog properties remained the same across the board, demonstrating a surprising ability to adapt to climatic variation. This is because species that were suited to a certain climate were replaced elsewhere by other species that were better suited to the new climate yet serving the same function within the ecosystem. The research has just been published in Nature Communications.

Peat bogs play an essential role on our planet. While they cover only 3% of the earth's surface, they store some 500 metric gigatons of carbon . This is the equivalent of 67% of all the CO2 in the air, or all the CO2 held in the world's boreal forestland, which makes up 10% of the earth's surface. A depth of one meter of peat corresponds to approximately 1,000 years of carbon storage. Because so much carbon is currently locked up in peat bogs, understanding how they respond to climate change is essential to know whether they might one day release massive amounts of CO2 into the air. Peat bogs are found on wet, acidic soil and have low biodiversity, which differs little from one region to the next - only around 60 species live in these ecosystems. What's more, these species are known to be sensitive to environmental conditions; they consist of vascular plants (shrubs, grasses and carnivorous plants) and moss (Sphagnum). Those factors would seem to indicate that peat bogs are exceptionally vulnerable to climate change, e.g., if the moss disappeared and vascular plants grew in its place, the ecosystem would shift dramatically and its CO2 storage capacity would shrink considerably.

Good news is hard to come by in environmental science these days. Fortunately, peat bogs' astonishing ability to resist climate change gives scientists reason to cheer. "Ecologists tend to believe that ecosystems with low biodiversity are more likely to disappear when environmental conditions change, even slightly. But what we found here opens the door to new ways of understanding biodiversity. Our study also underscores how important it is to preserve peat bogs - not just for their own sake, but also for the ecological services and benefits they bring," says Bragazza.

Analyzing 560 peat bog samples was no mean feat. The researchers first determined each sample's biodiversity "taxonomy," listing the number and abundance of each species in the samples. They then identified over a dozen functional traits of the species characterizing their role within its ecosystem. These functional characteristics include how they absorb water or capture light, the amount of carbon, nitrogen and phosphorous the moss contains, and the thickness, size and diameter of the vascular plants' leaves. The researchers obtained information about these functional characteristics from an existing database, and ran this information against the species they found in their peat bog samples. "We took the average value of each functional characteristic given in the database, factoring in the abundance of each species in the samples. That allowed us to go beyond simply comparing the species in our samples," says Bragazza.

Surprisingly, the researchers found "functional redundancy" in the samples. That is, the vascular plants and moss replaced each other based on the climatic conditions their ecosystem was exposed to, enabling the peat ecosystem as a whole to survive. "The average values for the functional characteristics didn't change, even if the taxonomy did. For instance, some species in the peat bog samples from Ireland don't exist in the samples from Sweden; those samples have other species that are better suited to the Swedish climate. But even when the species are different, they serve the same function within the ecosystem - they have the same size leaves and stems, for example," says Bragazza.

"Peat bogs are excellent CO2 sinks and have an unparalleled ability to adapt to climate change. That's in stark contrast to prairies, which nevertheless have greater biodiversity. Climate change can disrupt hay production in prairies and cause lasting damage to the ecosystem," says Buttler. Bragazza goes on to stress the importance of protecting peat bogs: "Their ability to adapt to climate change and therefore keep

on storing CO2 depends on how well their species can move from one bog to the next and create this 'functional redundancy.' Our study once again shows that it's vital to maintain peat bogs' biodiversity and prevent them from becoming isolated."

The vast amount of data that the team collected from peat bogs across Europe will be used to carry out further research, such as comparative analyses of the bogs' microbial compositions and assessments of their resistance thresholds. A promising way to learn more about peat bogs' unexpected properties.

Bjorn J. M. Robroek, Vincent E. J. Jassey, Richard J. Payne, Magalí Martí, Luca Bragazza, Albert Bleeker, Alexandre Buttler, Simon J. M. Caporn, Nancy B. Dise, Jens Kattge, Katarzyna Zaj?c, Bo H. Svensson, Jasper van Ruijven, and Jos T. A. Verhoeven. "Taxonomic and functional turnover are decoupled in European peat bogs," Nature Communications, 27 October 2017.

This study was carried out under the BiodivERsA-PEATBOG EU research program, with the support of several research institutes including EPFL and the Swiss Federal Institute for Forest, Snow and Landscape Research (WSL).

http://bbc.in/2xwcjJS

Marriage may protect against dementia

Marriage and having close friends may help protect against dementia, according to Loughborough University researchers.

The study, published in Journals of Gerontology, followed 6,677 adults for just under seven years. The quality of a person's social circle appeared more important than the overall size, the research team said. The Alzheimer's Society said it was essential to help patients to maintain "meaningful social connections".

None of the participants had dementia at the start of the trial, but 220 were diagnosed during it. The research group compared the traits of those who did and did not develop dementia to find clues as to how social lives affect risk. One finding was that when it comes to friends, it's quality, not quantity, that counts.

Prof Eef Hogervorst said: "You can be surrounded by people, but it is the number of close relationships that is associated with a reduced risk for dementia... it's not about the quantity." She thinks having close friends acts as a "buffer" against stress, which is linked to poor health.

Nine factors that contribute to dementia risk

Mid-life hearing loss - responsible for 9% of the risk
Failing to complete secondary education - 8%
Smoking - 5%
Failing to seek early treatment for depression - 4%
Physical inactivity - 3%
Social isolation - 2%
High blood pressure - 2%
Obesity - 1%
Type 2 diabetes - 1%

These risk factors - which are described as potentially modifiable - add up to 35%. The other 65% of dementia risk is thought to be potentially non-modifiable. The study also suggested that single people had twice the risk of developing dementia during the study than those who were married.

Dr Doug Brown, director of research at the Alzheimer's Society, said: "This amounts to about one extra diagnosis in each 100 unmarried people."

As the study only follows people over time it cannot prove cause and effect. Dementia is known to start in the brain decades before it is diagnosed and some of these early changes may affect people's ability to socialise.

Either way, Dr Brown said loneliness was a real issue in dementia. He said: "If people are not properly supported, dementia can be an incredibly isolating experience. "It is essential people with dementia are supported to maintain meaningful social connections and continue living their life as they want."