http://www.eurekalert.org/pub_releases/2014-07/bawh-ssg071714.php

Scientists successfully generate human platelets using next-generation bioreactor

Bioreactor-on-a-chip could help meet growing need for blood transfusions worldwide

Boston, MA - Scientists at Brigham and Women's Hospital (BWH) have developed a scalable, next-generation platelet bioreactor to generate fully functional human platelets in vitro. The work is a major biomedical advancement that will help address blood transfusion needs worldwide.

"The ability to generate an alternative source of functional human platelets with virtually no disease transmission represents a paradigm shift in how we collect platelets that may allow us meet the growing need for blood transfusions," said Jonathan Thon, PhD, Division of Hematology, BWH Department of Medicine, lead study author. The study is published July 21, 2014 in Blood.

According to the researchers, more than 2.17 million platelet units from donors are transfused yearly in the United States to treat patients undergoing chemotherapy, organ transplantation and surgery, as well as for those needing blood transfusions following a major trauma. However, increasing demand; a limited five-day shelf-life; and risk of contamination, rejection and infection have made blood platelet shortages common.

"Bioreactor-derived platelets theoretically have several advantages over conventional, donor-derived platelets in terms of safety and resource utilization," said William Savage, MD, PhD, medical director, Kraft Family Blood Donor Center at Dana Farber Cancer Institute/Brigham and Women's Hospital, who did not contribute to the study. "A major factor that has limited our ability to compare bioreactor platelets to donor platelets is the inefficiency of growing platelets, a problem that slows progress of clinical research. This study addresses that gap, while contributing to our understanding of platelet biology at the same time."

Blood cells, such as platelets, are made in bone marrow. The bioreactor - a device that mimics a biological environment to carry out a reaction on an industrial scale - uses biologically inspired engineering to fully integrate the major components of bone marrow, modeling both its composition and blood flow characteristics. The microfluidic platelet bioreactor recapitulates features such as bone marrow stiffness, extracellular matrix composition, micro-channel size, and blood flow stability under high-resolution live-cell microscopy to make human platelets. Application of shear forces of blood flow in the bioreactor triggered a dramatic increase in platelet initiation from 10 percent to 90 percent, leading to functional human platelets.

"By being able to develop a device that successfully models bone marrow represents a crucial bridge connecting our understanding of the physiological triggers of platelet formation to support drug development and scale platelet production," said senior study author Joseph Italiano, Jr., PhD, Division of Hematology, BWH Department of Medicine, and the Vascular Biology Program at Boston Children's Hospital.

In terms of next steps, the researchers would like to commence phase 0/I in human clinical trials in 2017. "The regulatory bar is appropriately set high for blood products, and it is important to us that we show platelet quality, function and safety over these next three years since we'll likely be recipients of these platelets ourselves at some point," said Thon.

This research was supported by the National Institutes of Health (R01Hl68130), American Society of Hematology Scholar Award, Brigham Research Institute at Brigham and Women's Hospital, and Marie Curie Actions International Outgoing Fellowship (300121).

Jonathan Thon, PhD, and Joseph Italiano, Jr., PhD are both founders of Platelet BioGenesis, a company that aims to produce donor-independent human platelets from human-induced pluripotent stem cells at scale.

http://www.medscape.com/viewarticle/828576

Sleep Deprivation Mimics Psychosis

After 24 hours of sleep deprivation, healthy individuals show symptoms of psychosis similar to those observed in schizophrenia, new research shows.

Nancy A. Melville

While underscoring the known adverse effects of severe insomnia on brain function, the study is the first to show sleep deprivation to trigger a key biomarker of psychosis that is important in the research of antipsychotic drugs - a reduction in prepulse inhibition of the acoustic startle response. "This strong main effect indicates that sleep deprivation might be an alternative method to the approach to pharmacologically induce deficits in prepulse inhibition in healthy volunteers," the authors write. The study was published July 2 in the *Journal of Neuroscience*.

Reliable Biomarker

Reduced prepulse inhibition is a reliable symptom not just of schizophrenia but also of schizophrenialike personality disorders, and it is seen in psychosis-prone healthy individuals. Specifically, it involves a reduced response to a strong stimulus, or pulse, if the stimulus is preceded with a weaker stimulus, or prepulse. Although the biomarker is often used in the research of antipsychotic compounds in animals, it is hard to mimic in humans without pharmacologic or experimental methods. "If the prepulse inhibition-decreasing effect of sleep deprivation can be replicated in humans, the sleep deprivation paradigm could prove a powerful model system of psychosis with strong clinical relevance," the authors write.

For the proof-of-concept study, lead author Ulrich Ettinger, MD, and colleagues with the Cognitive Psychology Unit, Department of Psychology, University of Bonn, Germany, evaluated acoustic prepulse inhibition and self-reported psychosislike symptoms in 24 healthy volunteers following a normal night's sleep and after a night of complete sleep deprivation. Participants were kept awake through the night with various activities, including conversation, movies, brief walks, and games.

After being kept up all night, participants were interviewed and were also assessed for prepulse inhibition, which involved exposure to a loud noise emitted through headphones and recording of the startle response with the use of electrodes to measure contraction in facial muscles.

Good Model of Schizophrenia

The results showed a robust effect in terms of significantly decreased prepulse inhibition associated with sleep deprivation ($P = .001$), and the severe insomnia also induced perceptual distortions, cognitive disorganization, and anhedonia (for all, $P < .02$). Importantly, sleep deprivation did not affect the degree or habituation of the startle response (for all, $P > .13$), indicating no effects of sleep deprivation on pulse only amplitudes.

The identification of a prepulse inhibition in relation to sleep deprivation is important because it represents a true symptom of psychosis that cannot be "faked," Dr. Ettinger told *Medscape Medical News.* "It's a cross-species phenomenon, and we already know a lot about it - for example, that it is impaired in schizophrenia, that it can be impaired in rats with ketamine/amphetamine, and that these impairments can be reversed with atypical antipsychotics."

"Thus, sleep deprivation may be a very good model of schizophrenia, in particular when combined with prepulse inhibition."

Sleep disturbances are common in people with schizophrenia, and severe insomnia is associated with exacerbations of the condition, leading to additional symptoms, but Dr. Ettinger said he was surprised to see the extent of effects of the loss of just 1 night of sleep even among healthy individuals.

"A single night of sleep deprivation may not at first seem like a particularly drastic intervention, and can for instance occur with students out partying or people working through the night; therefore, we were surprised to see such statistically significant increases in self-ratings of all 3 dimensions of schizophrenia - thought disorder, perceptual aberrations, and negative symptoms."

Out on a Limb?

Commenting on the study for *Medscape Medical News*, neurologist Donn Dexter, MD, said the effects of extreme insomnia have previously been associated with schizophrenialike symptoms.

"Prior research shows that prolonged sleep deprivation can actually cause a syndrome indistinguishable from paranoid schizophrenia. However, it is probably harder to get approval for such studies in current settings," said Dr. Dexter, a physician in the Neurology Department and Sleep Disorders Center at the Mayo Clinic Health System, in Eau Claire, Wisconsin.

Some important potential confounders, however, may limit the utility of the findings, he suggested. "The findings are interesting and, in a limited application, probably have real value, but I would be very careful about extending this too far," he said. "There are too many possible confounders, including that it's a small study with limited age groups, you're only looking at 1 biomarker and just 1 night of sleep deprivation, so it's kind of going out on a limb if you make too broad of a statement about it."

The authors and Dr. Dexter have disclosed no relevant financial relationships.

J Neurosci. 2014;34:9134-9140. Abstract

http://bit.ly/1ry2SSE

Giant Pterosaurs Serve as Aircraft Inspiration

Even the U.S. Department of Defense has shown interest in these long-extinct reptiles

Jul 15, 2014 |By Annie Sneed

Paleontologist Michael Habib studies the biomechanics of pterosaurs, the biggest of which - at 550 pounds and with a 34-foot wingspan - were the size of modern-day fighter jets. They were the largest flying animals ever to exist and sported anatomy different from any bird or bat. This makes them a unique model for flight mechanics, particularly for large aircraft.

To model how pterosaurs flew, Habib combines principles of physics and vertebrate anatomy with fossil data. He hopes that this knowledge will suggest new aircraft designs and other technology to places like nasa and the dod - it already has in some cases. In an abstract sense, he has brought these animals back from the dead. Pterosaur-inspired applications follow.

Flying Robots over Mars

Traditional spacecraft would need to fly extremely fast to stay aloft in Mars's thin atmosphere, an impracticality if scientists want to survey Martian terrain in detail. One solution may be a robot that flies like a pterosaur - with swift beating wings and a relatively slow-moving body. Hummingbirds and bumblebees also fly this way, and NASA has created designs for robots based on the biomechanics of these “flapping fliers.”

Morphing Wings

In each wing, pterosaurs had a single tapered finger that grew up to 2.5 meters long in the largest species. When pterosaurs flew, those fingers bent with the force

of the downward wing stroke and then reflexively snapped back into position on the upward stroke. The spontaneous return to equilibrium saved pterosaurs significant energy when flapping. Habib says roboticists in the U.S. Air Force are interested in morphing wings, which they could use in flight systems in aircraft or in parachutes - essentially highly convex wings.

Rapid-Launch Systems

Unlike planes today, giant pterosaurs did not need runways. They were experts at vertical takeoff, a feat that is impossible or incredibly inefficient for today's aircraft. Because the reptiles had stiff but lightweight, hollow bones, they could use all four limbs - both their feet and wings - to push powerfully against the ground. That action allowed them to generate more speed over a shorter distance as they leaped into flight. Habib is currently negotiating a Defense Advanced Research Projects Agency grant proposal with the DOD to design an aircraft system with analogous physical characteristics and a quadrupedal launch strategy that would allow pilots to perform a quick vertical launch or takeoff on low fuel.

Low-Flutter Tents

To fly, pterosaurs kept their wings uniformly taut. Those wings were membranous, with long, thick fibers crisscrossed by smaller fibers that controlled how much the wings fluttered. The fibers individually moved under high air pressure, but their varied dimensions meant they oscillated at opposing frequencies that ultimately canceled out, enabling pterosaurs to maintain a steady wing. Habib has approached manufacturers with a tent fabric design that exploits the same physical principle to reduce noisy flapping and improve stability in high wind conditions.

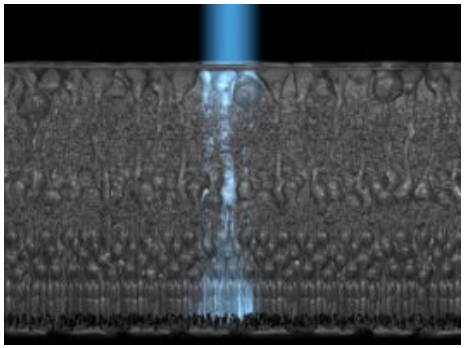
http://phys.org/news/2014-07-fiber-optic-pipes-retina-simple.html

Fiber optic light pipes in the retina do much more than simple image transfer

Having the photoreceptors at the back of the retina is not a design constraint, it is a design feature.

Phys.org - The idea that the vertebrate eye, like a traditional front-illuminated camera, might have been improved somehow if it had only been able to orient its wiring behind the photoreceptor layer, like a cephalopod, is folly. Indeed in simply engineered systems, like CMOS or CCD image sensors, a back-illuminated design manufactured by flipping the silicon wafer and thinning it so that light hits the photocathode without having to navigate the wiring layer can improve photon capture across a wide wavelength band. But real eyes are much more crafty than that.

A case in point are the Müller glia cells that span the thickness of the retina. These high refractive index cells spread an absorptive canopy across the retinal surface and then shepherd photons through a low-scattering cytoplasm to separate receivers, much like coins through a change sorting machine. A new paper in Nature Communications describes how these wavelength-dependent wave-guides can shuttle green-red light to cones while passing the blue-purples to adjacent rods. The idea that these Müller cells act as living fiber optic cables has been floated previously. It has even been convincingly demonstrated using a dual beam laser trap. In THIS case (THIS, like in Java programming meaning the paper just brought up) the authors couched this feat as mere image transfer, with the goal just being to bring light in with minimal distortion.



Muller Cells appear to act as living optical fibers. **vision-research.eu**

Fireflies, in trying to get light through their cuticle, face a similar but opposite challenge - namely, getting light out. Their fascinating solutions to transparency and index matching are an illuminating read. In the retina, and indeed the larger light organ that is the eye, there is much more going on than just photons striking rhodopsin photopigments. As far as absorbers, there are all kinds of things going on in there - various carontenoids, lipofuscins and lipochromes, even cytochrome oxidases in mitochondria that get involved at the longer wavelegnths. Speaking of the mitochondria, one of their most incredible adaptions in the eye came to my attention recently courtesy of O.R. Pagan, author of a cool book about planarians. His blog mentions how these creatures have convinced the endosymbiont microbes in their eyes to accumulate refractive proteins and tightly pack together. After swelling to several times normal size like a liver about to become foie gras, these mitochondria are transformed into a lens about to focus light onto sensitive cells.

In considering not just the classical photoreceptors but the entire retina itself as a light-harvesting engine, it seems prudent to also regard its entire synaptic endowment as a molecular-scale computing volume. In other words, when you have many cells that have no axons or spikes to speak of, that can completely refigure their fine structure within a few minutes to handle changing light levels, every synapse appears as an essential machine that percolates information as if at the Brownian scale, or even below.

By contrast the brain itself, while containing much the same, appears not quite so tightly strung. That's not to say that wiping out swaths of cherished synaptic meat memory in the brain would be on par with taking down a few tubules of kidney,

lobules of liver, or osteons of bone, it's just the retina seems even more brain-like than the brain itself. The retinas of different animals clearly employ different tricks. Some reflect incoming light back out through the retina for a second look. Others can detect things like polarization or even angle of incidence.

Most incredibly, like the wings of a swallow, the retina more-or-less works right out of the box, even if it has not seen any exercise. In seeking to understand how it then further refines its delicate structure we should perhaps not overlook the pervasive organizing influence of the incoming photons themselves. Now that it is becoming abundantly clear that the whole works can "feel" them, the next question to answer is how.

More information: Müller cells separate between wavelengths to improve day vision with minimal effect upon night vision, Nature Communications 5, Article number: 4319 DOI: 10.1038/ncomms5319

Abstract

Vision starts with the absorption of light by the retinal photoreceptors - cones and rods. However, due to the 'inverted' structure of the retina, the incident light must propagate through reflecting and scattering cellular layers before reaching the photoreceptors. It has been recently suggested that Müller cells function as optical fibres in the retina, transferring light illuminating the retinal surface onto the cone photoreceptors. Here we show that Müller cells are wavelength-dependent wave-guides, concentrating the green-red part of the visible spectrum onto cones and allowing the blue-purple part to leak onto nearby rods. This phenomenon is observed in the isolated retina and explained by a computational model, for the guinea pig and the human parafoveal retina. Therefore, light propagation by Müller cells through the retina can be considered as an integral part of the first step in the visual process, increasing photon absorption by cones while minimally affecting rod-mediated vision.

http://www.eurekalert.org/pub_releases/2014-07/ifhm-dai071714.php

Deaths and infections from HIV, tuberculosis, and malaria plummet globally

New HIV infections dropped by almost one-third from the epidemic peak; TB deaths declined by 3.7 percent between 2000 and 2013; child deaths from malaria in sub-Saharan Africa have dropped 31.5 percent in the past decade

SEATTLE -Today, fewer people are dying from HIV/AIDS, tuberculosis, and malaria, according to a new, first-of-its-kind analysis of trend data from 188 countries. The pace of decline in deaths and infections has accelerated since 2000, when the Millennium Development Goals (MDGs) were established to stop the spread of these diseases by 2015.

HIV interventions - including antiretroviral therapy (ART), prevention of mother to child transmission (PMTCT), and HIV prophylaxis - have been successful. HIV is increasingly a condition people live with rather than die from, and the world has added nearly 20 million life years as a result of these programs.

About 70% of those years of life were in the developing world. In terms of age, 14% of the years of life saved were in children under age 15, 50% were in 15- to 49-year-olds, and 36% were in people age 50 and over. But despite considerable progress, more must be done to reduce deaths and infections further.

In the case of HIV, researchers note that the comparatively low price per year of life saved is one of the major achievements in global health in the past decade. Comparison of the total amount invested in HIV prevention and treatment to the years of life saved during 2000 - 11 yields in developing countries a ratio of $4498 per life-year saved. In 2011, all donors combined spent US$7.7 billion on HIV/AIDS.

Published in The Lancet on July 22, the study, "Global, regional, and national incidence and mortality for HIV, tuberculosis, and malaria during 1990 - 2013: a systematic analysis for the Global Burden of Disease Study 2013," was conducted by an international consortium of researchers led by the Institute for Health Metrics and Evaluation (IHME) at the University of Washington.

The findings were released at the International AIDS Conference in Melbourne. Dr. Christopher Murray, director of IHME and a co-founder of the Global Burden of Disease (GBD) study, presented the findings at an event where he was joined by Richard Horton, Editor-in-Chief of The Lancet; Michel Sidibé, Executive Director of UNAIDS; Deborah Birx, United States Global AIDS Coordinator, PEPFAR; and Mark Dybul, Executive Director, Global Fund to Fight AIDS, Tuberculosis and Malaria.

"The global investment in HIV treatment is saving lives at a rapid clip," said Dr. Murray. "But the quality of antiretroviral programs varies widely. In order to reduce HIV-related deaths even further, we need to learn from the best programs and do away with the worst ones."

Researchers found that greater access to treatment is needed as well. Globally, in 2013, there were nearly 30 million people living with HIV, 1.8 million new infections, and 1.3 million deaths from the disease. At the peak of the epidemic in 2005, HIV caused 1.7 million deaths. Global HIV incidence peaked in 1997 with 2.8 million new infections and has declined since the peak at 2.7% per year.

The study reveals substantial changes to previous understanding of HIV epidemics. In Latin America and Eastern Europe, HIV epidemics are substantially smaller than previously estimated - while in some countries, like the Philippines, the crisis is actually much bigger.

This updated analysis shows some notable differences when compared to the GBD 2010 study and new estimates were found to be lower than previous

estimates. Estimated HIV deaths in Latin American countries such as Peru, Venezuela, and Colombia were reduced by more than two-thirds in the year 2010. In the same year, the population of people living with HIV in Eastern and Central Europe was found to be over 60% smaller than previously estimated, highlighted by a 69% decrease in Russia.

Improvements in IHME's methodology revealed that cumulatively, countries identified as having concentrated epidemics had 39% fewer deaths and 53% fewer people living with HIV. In contrast, deaths in countries with generalized epidemics increased by 23%, and the HIV-infected populations were slightly higher by 3%.

Success in reducing HIV has had an impact on tuberculosis as well. Since 2000, global progress in reducing TB prevalence and mortality has accelerated. Prevalence rates rose slightly between 1990 and 2000 at an annual rate of 0.4% but declined by 1.3% per year from 2000 to 2013.

TB rates globally have declined between 2000 and 2013, due largely to progress in two regions: East and South Asia. In South Asia, which accounts for almost half of TB deaths, mortality rates declined annually by 4.2% during this period. In East Asia, death rates fell annually by 7.5%. In 2013, there were 7.5 million new TB cases, and the disease caused 1.4 million deaths worldwide.

Earlier and more effective treatment has helped shorten the duration of TB infections, but the authors note that as the world ages, higher numbers of cases and deaths will occur. When looking at death rates that are age-standardized, meaning they were adjusted for differences in population size and ages over time and across countries, the countries within Latin America and the Caribbean with the highest TB rates are Bolivia, Peru, and Haiti. The countries with the highest death rates from TB are in sub-Saharan Africa: Somalia, Central African Republic, South Sudan, Zambia, and Mozambique.

The most significant reductions in TB deaths - declining at a rate of 3.7% between 2000 and 2013 - occurred among people who are HIV-negative. Men and boys make up the majority of TB cases among people who are HIV-negative and die at higher rates (64.7%) than HIV-negative women and girls with TB. In 2013, 83.2% of cases and 58.8% of deaths in HIV-negative people with TB occurred under age 60. °"As the world's population grows older, tuberculosis will remain a major health threat," said Dr. Nobhojit Roy of the BARC Hospital in India and a co-author of the study. "TB presents unique challenges across different countries and regions, and better data will help drive the most effective strategies to address it."

Similar to HIV and TB, researchers found that an increased focus due to the Millennium Development Goals has helped drive down malaria infections and deaths.

Globally, malaria cases and deaths grew rapidly from 1990, reaching a peak of 232 million cases in 2003 and 1.2 million deaths in 2004. As with HIV, the burden of malaria is concentrated in sub-Saharan Africa. Two of the three countries - Nigeria, Democratic Republic of the Congo (DRC), and India - that accounted for roughly half of all malaria deaths in 2013 are in Africa. DRC is also among four countries, three of them in Africa, that have more than 5 million malaria cases a year. DRC and Mozambique both have 6 million, Nigeria has 30 million, and India has more than 60 million.

Since 2004, child deaths from malaria in sub-Saharan Africa have declined 31.5%. Since 2000, the vast majority of countries - including those in sub-Saharan Africa - saw declines in age-standardized malaria death rates. Annual malaria deaths began to decline from a peak of 1.2 million in 2004 to about 855,000 in 2013, having increased from 888,000 in 1990. The highest rates of age-standardized malaria deaths were in Mozambique, Burkina Faso, Guinea-Bissau, Mali, Sierra Leone, The Gambia, and Guinea.

Outside of Africa, malaria mortality has been steadily declining since 1990 as well, but Yemen, India, Myanmar, and Papua New Guinea all have malaria death rates over 7.5 per 100,000. By contrast, certain countries in Southeast Asia (Thailand and Malaysia) have achieved very low death rates.

"Great progress has been made in reducing malaria deaths and infections, but we need more success stories throughout Africa in particular for us to eliminate malaria," said Dr. Corine Karema, of the Malaria & Other Parasitic Diseases Division, Ministry of Health Rwanda, and a co-author of the study. "Malaria is notoriously difficult to early diagnose, treat promptly using efficacious drugs, and track, and part of the strategy in fighting it is to invest in gathering better evidence through a robust surveillance system."

With progress in reducing HIV at the global level, success in particular countries and regions varies as the HIV epidemic has peaked and declined at different times. Regionally, the burden of HIV is concentrated in sub-Saharan Africa. Prevalence levels are highest in Botswana, Lesotho, and Swaziland (above 12,000 per 100,000 people). HIV rates in Botswana, for example, are 15 times higher than in the DRC and 40 times higher than Niger.

Researchers found similar variation in other regions. In Southeast Asia, HIV rates are substantially higher in Thailand and Papua New Guinea. HIV rates are relatively high in parts of Europe and Central Asia (Portugal, Spain, Ukraine, Russia, and Kazakhstan) and in Latin America and the Caribbean (Panama, Honduras, Belize, Guatemala, Guyana, Suriname, Haiti, Dominican Republic, Jamaica, and the Bahamas), where prevalence levels exceed 220 per 100,000.

The annual number of new infections has declined by almost one-third between the global peak in 1997 and 2013. New infections in children have decreased by more than 7% each year since 2000, compared to a 2.4% annual decrease in adults - demonstrating the impact of interventions in reducing transmission between mothers and their children.

New infections in children declined from 340,000 in 2000 to 134,000 in 2013, at an annual rate of 7.2%, while new infections in adults declined from 2.3 million to 1.7 million, falling at 2.4% per year, on average, over this period.

Great progress has been achieved in reducing HIV infection in children (62.4% reduction since the incidence peak in 2002) due to the scale-up of interventions. However, the continued 1.7 million new infections per year in adults, while down 32.7% from the peak of the epidemic at the global scale, is a stark reminder of the continuing epidemic.

In 2013, new cases of HIV occurred equally in men and women, and HIV incidence in children as well as older adults is similar for both genders. However, there were more infections in women than there were for men at ages 15-24 years, and more HIV deaths occur in males (53.9%) than in women and girls.

"This massive new study, on the eve of the end of the MDG era, documents impressive recent progress against HIV and malaria, in particular, but it also shows that much more needs to be done. HIV, TB, and malaria each currently cause about 1 million deaths a year," said Dr. Alan Lopez, Melbourne Laureate Professor at the University of Melbourne and co-founder of the GBD study. "All three are major causes of health loss in poor countries, and all three should be a key focus of concerted global health action and support. Without it, we risk stagnation, or even worse, unconscionable reversal of recent gains."

Download the study at: http://press.thelancet.com/GBDMDG6.pdf

http://www.eurekalert.org/pub_releases/2014-07/miot-tta072114.php

Try, try again? Study says no

When learning certain elements of language, adults' more highly developed cognitive skills actually get in the way

CAMBRIDGE, MA - When it comes to learning languages, adults and children have different strengths. Adults excel at absorbing the vocabulary needed to navigate a grocery store or order food in a restaurant, but children have an uncanny ability to pick up on subtle nuances of language that often elude adults. Within months of living in a foreign country, a young child may speak a second language like a native speaker.

Brain structure plays an important role in this "sensitive period" for learning language, which is believed to end around adolescence. The young brain is equipped with neural circuits that can analyze sounds and build a coherent set of rules for constructing words and sentences out of those sounds. Once these language structures are established, it's difficult to build another one for a new language.

In a new study, a team of neuroscientists and psychologists led by Amy Finn, a postdoc at MIT's McGovern Institute for Brain Research, has found evidence for another factor that contributes to adults' language difficulties: When learning certain elements of language, adults' more highly developed cognitive skills actually get in the way. The researchers discovered that the harder adults tried to learn an artificial language, the worse they were at deciphering the language's morphology - the structure and deployment of linguistic units such as root words, suffixes, and prefixes.

"We found that effort helps you in most situations, for things like figuring out what the units of language that you need to know are, and basic ordering of elements. But when trying to learn morphology, at least in this artificial language we created, it's actually worse when you try," Finn says.

Finn and colleagues from the University of California at Santa Barbara, Stanford University, and the University of British Columbia describe their findings in the July 21 issue of PLOS ONE. Carla Hudson Kam, an associate professor of linguistics at British Columbia, is the paper's senior author.

Too much brainpower

Linguists have known for decades that children are skilled at absorbing certain tricky elements of language, such as irregular past participles (examples of which, in English, include "gone" and "been") or complicated verb tenses like the subjunctive. "Children will ultimately perform better than adults in terms of their command of the grammar and the structural components of language - some of the more idiosyncratic, difficult-to-articulate aspects of language that even most native speakers don't have conscious awareness of," Finn says.

In 1990, linguist Elissa Newport hypothesized that adults have trouble learning those nuances because they try to analyze too much information at once. Adults have a much more highly developed prefrontal cortex than children, and they tend to throw all of that brainpower at learning a second language. This high-powered processing may actually interfere with certain elements of learning language. "It's an idea that's been around for a long time, but there hasn't been any data that experimentally show that it's true," Finn says.

Finn and her colleagues designed an experiment to test whether exerting more effort would help or hinder success. First, they created nine nonsense words, each with two syllables. Each word fell into one of three categories (A, B, and C), defined by the order of consonant and vowel sounds.

Study subjects listened to the artificial language for about 10 minutes. One group of subjects was told not to overanalyze what they heard, but not to tune it out either. To help them not overthink the language, they were given the option of completing a puzzle or coloring while they listened. The other group was told to try to identify the words they were hearing.

Each group heard the same recording, which was a series of three-word sequences - first a word from category A, then one from category B, then category C - with no pauses between words. Previous studies have shown that adults, babies, and even monkeys can parse this kind of information into word units, a task known as word segmentation.

Subjects from both groups were successful at word segmentation, although the group that tried harder performed a little better. Both groups also performed well in a task called word ordering, which required subjects to choose between a correct word sequence (ABC) and an incorrect sequence (such as ACB) of words they had previously heard.

The final test measured skill in identifying the language's morphology. The researchers played a three-word sequence that included a word the subjects had not heard before, but which fit into one of the three categories. When asked to judge whether this new word was in the correct location, the subjects who had been asked to pay closer attention to the original word stream performed much worse than those who had listened more passively.

Turning off effort

The findings support a theory of language acquisition that suggests that some parts of language are learned through procedural memory, while others are learned through declarative memory. Under this theory, declarative memory, which stores knowledge and facts, would be more useful for learning vocabulary and certain rules of grammar. Procedural memory, which guides tasks we perform without conscious awareness of how we learned them, would be more useful for learning subtle rules related to language morphology.

"It's likely to be the procedural memory system that's really important for learning these difficult morphological aspects of language. In fact, when you use the declarative memory system, it doesn't help you, it harms you," Finn says.

Still unresolved is the question of whether adults can overcome this language-learning obstacle. Finn says she does not have a good answer yet but she is now testing the effects of "turning off" the adult prefrontal cortex using a technique called transcranial magnetic stimulation. Other interventions she plans to study include distracting the prefrontal cortex by forcing it to perform other tasks while language is heard, and treating subjects with drugs that impair activity in that brain region.

The research was funded by the National Institute of Child Health and Human Development and the National Science Foundation.

http://www.eurekalert.org/pub_releases/2014-07/tuhs-tur071614.php

Temple University researchers eliminate the HIV virus from cultured human cells for first time

Their approach promises a permanent cure and potential for protection against HIV

Philadelphia, PA - The HIV-1 virus has proved to be tenacious, inserting its genome permanently into its victims' DNA, forcing patients to take a lifelong drug regimen to control the virus and prevent a fresh attack. Now, a team of Temple University School of Medicine researchers has designed a way to snip out the integrated HIV-1 genes for good.

"This is one important step on the path toward a permanent cure for AIDS," says Kamel Khalili, PhD, Professor and Chair of the Department of Neuroscience at Temple. Khalili and his colleague, Wenhui Hu, MD, PhD, Associate Professor of Neuroscience at Temple, led the work which marks the first successful attempt to eliminate latent HIV-1 virus from human cells.

"It's an exciting discovery, but it's not yet ready to go into the clinic. It's a proof of concept that we're moving in the right direction," added Dr. Khalili, who is also Director of the Center for Neurovirology and Director of the Comprehensive NeuroAIDS Center at Temple.

In a study published July 21 by the Proceedings of the National Academy of Sciences, Khalili and colleagues detail how they created molecular tools to delete the HIV-1 proviral DNA. When deployed, a combination of a DNA-snipping enzyme called a nuclease and a targeting strand of RNA called a guide RNA (gRNA) hunt down the viral genome and excise the HIV-1 DNA. From there, the cell's gene repair machinery takes over, soldering the loose ends of the genome back together – resulting in virus-free cells.

"Since HIV-1 is never cleared by the immune system, removal of the virus is required in order to cure the disease," says Khalili, whose research focuses on the neuropathogenesis of viral infections. The same technique could theoretically be used against a variety of viruses, he says. The research shows that these molecular tools also hold promise as a therapeutic vaccine; cells armed with the nuclease-RNA combination proved impervious to HIV infection.

Worldwide, more than 33 million people have HIV, including more than 1 million in the United States. Every year, another 50,000 Americans contract the virus, according to the U.S. Centers for Disease Control and Prevention.

Although highly active antiretroviral therapy (HAART) has controlled HIV-1 for infected people in the developed world over the last 15 years, the virus can rage

again with any interruption in treatment. Even when HIV-1 replication is well controlled with HAART, the lingering HIV-1 presence has health consequences. "The low level replication of HIV-1 makes patients more likely to suffer from diseases usually associated with aging," Khalili says. These include cardiomyopathy – a weakening of the heart muscle – bone disease, kidney disease, and neurocognitive disorders. "These problems are often exacerbated by the toxic drugs that must be taken to control the virus," Khalili adds.

Researchers based the two-part HIV-1 editor on a system that evolved as a bacterial defense mechanism to protect against infection, Khalili says. Khalili's lab engineered a 20-nucleotide strand of gRNA to target the HIV-1 DNA and paired it with Cas9. The gRNA targets the control region of the gene called the long terminal repeat (LTR). LTRs are present on both ends of the HIV-1 genome. By targeting both LTRs, the Cas9 nuclease can snip out the 9,709-nucleotides that comprise the HIV-1 genome. To avoid any risk of the gRNA accidentally binding with any part of the patient's genome, the researchers selected nucleotide sequences that do not appear in any coding sequences of human DNA, thereby avoiding off-target effects and subsequent cellular DNA damage.

The editing process was successful in several cell types that can harbor HIV-1, including microglia and macrophages, as well as in T-lymphocytes. "T-cells and monocytic cells are the main cell types infected by HIV-1, so they are the most important targets for this technology," Khalili says.

The HIV-1 eradication approach faces several significant challenges before the technique is ready for patients, Khalili says. The researchers must devise a method to deliver the therapeutic agent to every single infected cell. Finally, because HIV-1 is prone to mutations, treatment may need to be individualized for each patient's unique viral sequences.

"We are working on a number of strategies so we can take the construct into preclinical studies," Khalili says. "We want to eradicate every single copy of HIV-1 from the patient. That will cure AIDS. I think this technology is the way we can do it."

In addition to Khalili and Hu, the other authors of the PNAS paper are Rafal Kaminski, Fan Yang, Yonggang Zhang, of Temple's Department of Neuroscience; Biao Luo of the Cancer Genome Institute, Fox Chase Cancer Center, Temple University School of Medicine; Jonathan Karn, David Alvarez-Carbonell, Yoelvis Garcia, of the Department of Molecular Biology and Microbiology, Case Western Reserve University, Cleveland; and Xianming Mo of the Laboratory of Stem Cell Biology in the West China Medical School, Sichuan University, Chengdu, China.

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http://www.scientificamerican.com/article/cystic-fibrosis-might-be-2-diseases/

Cystic Fibrosis Might Be 2 Diseases

The sister disease affects the pancreas and other organs, while leaving the lungs alone

Jul 21, 2014 |By Beth Skwarecki

Thick mucus that can drown the lungs of a child has long been the hallmark of cystic fibrosis. The hereditary disease affects 30,000 Americans, and patients die unless they receive treatment to clear their lungs.

But new research suggests that this pulmonary view of cystic fibrosis is only half of the picture: a suite of symptoms associated with cystic fibrosis can also occur in patients who do not have lung disease at all, indicating that cystic fibrosis is really two diseases.

This second version, it appears, causes pancreatitis.

"Cystic fibrosis has been evaluated and managed by pulmonary doctors focusing on the lung, but other important problems are never seen by the pulmonologist and nobody's put the pieces together," says David Whitcomb of the University of Pittsburgh, who studies disorders of the pancreas.

Cystic fibrosis results from mutations in a gene that produces a tube-shaped protein known as CFTR, essential to the balance of electrolytes in the body. Specifically, this protein allows chloride ions to pass in and out of cells. When it malfunctions in classic cystic fibrosis, cells in the airway cannot produce normal mucus but instead make a thicker, stickier substance that clogs the lungs.

But CFTR leads a double life.

Whitcomb's team screened a group of nearly 1,000 patients with pancreatitis and found nine abnormal but supposedly harmless versions of the CFTR gene.

Their study suggests that the seemingly benign mutations break the switch that turns CFTR from a chloride portal to a channel for bicarbonate, a chemical that the pancreas produces to neutralize stomach acid.

Patients with these mutations do not have the problems associated with the chloride channel, but the faulty bicarbonate channel means that they can suffer from painful pancreatitis, as well as sinusitis and, in men, infertility.

Computer simulations confirmed that the mutations are all in places that would inhibit bicarbonate but not chloride from passing through.

Without the ability to secrete bicarbonate, Whitcomb says, patients cannot flush digestive enzymes out of their pancreas and the pancreas essentially dissolves itself, a horrifically painful condition.

Other organs also depend on the bicarbonate channel: cells in the sinuses use it to produce the right consistency of mucus, and it is essential for pH-balancing semen.

The CFTR mutations that only affect bicarbonate thus cause a recognizable syndrome that combines the symptoms of pancreatitis, sinusitis and male infertility. Meanwhile, since the chloride-control function of the channel is unaffected, these patients pass the sweat chloride test - the standard for diagnosing cystic fibrosis - with flying colors.

Drugs that help cystic fibrosis patients may also relieve this form of pancreatitis. If the number of patients with the syndrome turns out to be large - Whitcomb suspects it is more than the number with classic cystic fibrosis - then the market for those drugs will grow, and the drugs themselves could become more affordable. The cystic fibrosis drug Kalydeco, for example, currently costs more than $300,000 per year.

Julie Forman-Kay, a biochemist at the Hospital for Sick Children in Toronto, notes that the techniques the researchers used to figure out the details of how each mutation changes the protein are "extremely challenging" and "kind of an art form," and more work is needed to confirm that the mutations actually cause the changes that the computer model predicts.

"The real potential impact of this paper," she says, "is that it kind of wakes up the research community a bit and pushes for expanding understanding of the role of CFTR in other diseases besides cystic fibrosis."

In a sense, cystic fibrosis is returning to its roots. It was originally named "cystic fibrosis of the pancreas," and children with the disease experienced pancreatic failure in infancy and died within their first few years of life.

Once pills were available to replace the digestive enzymes they lost, those children were able to grow up - the average life expectancy is now 40 years - and the focus of treatment shifted to the lungs.

Whitcomb's eventual goal is to disentangle the distinct causes of what, until recently, appeared to be a single disease. "Chronic pancreatitis was considered to be a total enigma," Whitcomb says, with 42 percent of cases having no known cause, "but we're finding that it's five or six or more different diseases that all look the same on CAT scans. What we're able to do now is unravel that mystery on a case-by-case basis."

CFTR-caused pancreatitis is one of those variants. Another, which Whitcomb's group identified over a decade ago, is caused when a digestive enzyme, trypsin, is activated at the wrong time and digests the pancreas from within.

"Modern medicine is built on the germ theory of disease, that one factor will cause a complex disorder," Whitcomb says, but personalized medicine is showing that many disorders have different causes in different patients.

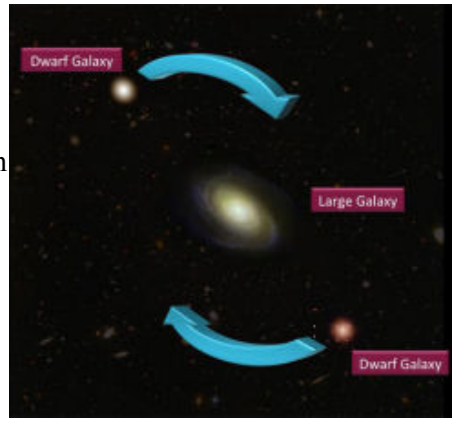
As the differences are untangled, Whitcomb says, "the percentage of patients that are mystery patients is getting smaller and smaller."

http://bit.ly/1pRGEYR

Dwarf Galaxy Movement Challenges to Our Understanding the Universe

New Study on Dwarf Galaxies May Force a Cosmic Rethink

A newly published study examines the movement of small galaxies throughout the universe, finding that they 'dance' in orderly disc-shaped orbits around larger galaxies.



The discovery that many small galaxies throughout the universe do not 'swarm' around larger ones like bees do but 'dance' in orderly disc-shaped orbits is a challenge to our understanding of how the universe formed and evolved. The finding, by an international team of astronomers, including Professor Geraint Lewis from the University of Sydney's School of Physics, is announced in the journal Nature.

This is an artist's impression of the coherent orbit of dwarf galaxies about a large galaxy. **Geraint Lewis.**

"Early in 2013 we announced our startling discovery that half of the dwarf galaxies surrounding the Andromeda Galaxy are orbiting it in an immense plane" said Professor Lewis. "This plane is more than a million light years in diameter, but is very thin, with a width of only 300,000 light years."

The universe contains billions of galaxies. Some, such as the Milky Way, are immense, containing hundreds of billions of stars. Most galaxies, however, are dwarfs, much smaller and with only a few billion stars.

For decades astronomers have used computer models to predict how these dwarf galaxies should orbit large galaxies. They had always found that they should be scattered randomly. "Our Andromeda discovery did not agree with expectations, and we felt compelled to explore if it was true of other galaxies throughout the universe," said Professor Lewis.

Using the Sloan Digital Sky Survey, a remarkable resource of color images and 3-D maps covering more than a third of the sky, the researchers dissected the properties of thousands of nearby galaxies. "We were surprised to find that a large proportion of pairs of satellite galaxies have oppositely directed velocities if they are situated on opposite sides of their giant galaxy hosts", said lead author Neil Ibata of the Lycée International in Strasbourg, France.

"Everywhere we looked we saw this strangely coherent coordinated motion of dwarf galaxies. From this we can extrapolate that these circular planes of dancing dwarfs are universal, seen in about 50 percent of galaxies," said Professor Geraint Lewis. "This is a big problem that contradicts our standard cosmological models. It challenges our understanding of how the universe works including the nature of dark matter."

The researchers believe the answer may be hidden in some currently unknown physical process that governs how gas flows in the universe, although, as yet, there is no obvious mechanism that can guide dwarf galaxies into narrow planes. Some experts, however, have made more radical suggestions, including bending and twisting the laws of gravity and motion. "Throwing out seemingly established laws of physics is unpalatable," said Professor Lewis, "but if our observations of nature are pointing us in this direction, we have to keep an open mind. That's what science is all about."

Publication: Neil G. Ibata, et al., "Velocity anti-correlation of diametrically opposed galaxy satellites in the low-redshift Universe," Nature, 2014; doi:10.1038/nature13481

http://bit.ly/1rFeZPA

Oceans Make Exoplanets Stable for Alien Life

The role that Earth's oceans have on our planet's habitability is undeniable, but now scientists think that exoplanetary oceans are essential for alien life to evolve.

Jul 21, 2014 01:49 PM ET // by Ian O'Neill

In a new study published by the journal Astrobiology, University of East Anglia (UEA), UK, researchers have come to the conclusion that, to make a planet habitable, a large liquid ocean is needed to stabilize its atmosphere.

"We know that many planets are completely uninhabitable because they are either too close or too far from their sun," said David Stevens of UEA's School of Maths. "A planet's habitable zone is based on its distance from the sun and temperatures at which it is possible for the planet to have liquid water. "But until now, most habitability models have neglected the impact of oceans on climate."

The habitable zone surrounding any star is the distance at which it's not too hot and not too cold to support liquid water on a planetary surface. Liquid water is vital for the evolution of life as we know it.

Earth orbits within our sun's habitable zone, unsurprisingly, whereas Mars is located on the outside edge and Venus on the inside edge. The life-giving contrast between Earth, Mars and Venus couldn't be more stark; Mars is a frozen, dry wasteland with dramatic surface temperature variations, and Venus is a choked, broiling world with searing surface temperatures. But Earth is stable, a factor that has allowed life to thrive for billions of years.

Although a planet's distance from its star is important, whether or not it has an ocean appears to be a huge factor. In fact, the presence of an ocean is the ultimate planetary "climate control" for any planet, according to new computer models created by Stevens' team.

"Oceans have an immense capacity to control climate," he said in a UEA news release. "They are beneficial because they cause the surface temperature to respond very slowly to seasonal changes in solar heating. And they help ensure that temperature swings across a planet are kept to tolerable levels."

Although Mars is located on the outside edge of the sun's habitable zone, planetary scientists believe the red planet once possessed large bodies of water when the planet's atmosphere was thicker. The presence of liquid water on the surface of ancient Mars is exciting - after all, on Earth, where there's water there's usually life. But the presence of possible Martian oceans may have stabilized the atmosphere, making it less prone to wild temperature fluctuations and more comfortable for life to gain a foothold. Modern Mars endures air temperature fluctuations of over 100 degrees Celsius (212 degrees Fahrenheit).

In the computer model, the researchers found that the heat transported by a global ocean has a "major impact on the temperature distribution across a planet," said Stevens. This, he argues, would make larger regions of an ocean-supporting exoplanet habitable.

"Oceans help to make a planet's climate more stable so factoring them into climate models is vital for knowing whether the planet could develop and sustain life," added Stevens. "This new model will help us to understand what the climates of other planets might be like with more accurate detail than ever before."

This study once again proves that while finding an exoplanet orbiting within its star's habitable zone is important, the real "holy grail" for finding a truly habitable world would be to look for rocky exoplanets possessing global oceans, worlds that may be more abundant than we thought.

http://www.bbc.com/news/health-28401693

'Eighty new genes linked to schizophrenia'

Scientists have uncovered 80 previously unknown genes which may put people at risk of developing schizophrenia, research in Nature suggests.

By Smitha Mundasad Health reporter, BBC News

The team says the world's largest genetic study of the disease shows it can have biological causes - putting it on a par with other medical conditions. Led by Cardiff University, the international group believes this could be a launch pad for new therapies. Charities say that holistic approaches to the illness must continue.

Scientists have debated the relative role genes play in schizophrenia - a condition which affects more than 24 million people worldwide - for many years.

Now a global consortium across 35 countries has examined the genetic make-up of more than 37,000 people with the condition, comparing them with some 110,000 people without the disease.

'New biology'

Scientists found more than 100 genes that make people more susceptible to schizophrenia- 83 of which have never been pinpointed before. Many of these genes are involved in the relay of chemical messages around the brain. And others are known to be involved in the immune system - affecting the body's natural armoury against disease.

Prof Michael O'Donovan of Cardiff University who led the research said: "For many years it has been difficult to develop new lines of treatment for schizophrenia, hampered by a poor understanding of the biology of disease. "Finding a whole new bunch of genetic associations opens a window for well-informed experiments to unlock the biology of this condition and we hope ultimately new treatments."

Prof David Curtis of University College London and one of the authors of the research told the BBC: "This study puts psychiatry into the same category as other parts of medicine. "In the past we have struggled with the view that psychiatric conditions are not 'real' illnesses but early genetic studies had limited successes. "Now we show with confidence that there are biological processes going awry."

Dr Gerome Breen of King's College London who was not involved in the current research but will be working on future studies told the BBC: "I think this is revolutionary. "We now have a massive amount of new biology to investigate - a whole new set of ideas which could provide many potential avenues for treatment. "This is crucial. Drug therapy for schizophrenia has not changed significantly since the 1970s."

Promising step

Beth Murphy at the charity Mind said:"Today's research provides an interesting and promising step in the search for more suitable treatment options than are currently available. "But it is vital that health professionals recognise the need for a holistic approach in treating those who are experiencing schizophrenia and bipolar disorder right now."

http://www.eurekalert.org/pub_releases/2014-07/uoi-rcv072214.php

Researchers create vaccine for dust-mite allergies

If you're allergic to dust mites (and chances are you are), help may be on the way.

Researchers at the University of Iowa have developed a vaccine that can combat dust-mite allergies by naturally switching the body's immune response. In animal tests, the nano-sized vaccine package lowered lung inflammation by 83 percent despite repeated exposure to the allergens, according to the paper, published in the AAPS (American Association of Pharmaceutical Scientists) Journal. One big reason why it works, the researchers contend, is because the vaccine package contains a booster that alters the body's inflammatory response to dust-mite allergens.

"What is new about this is we have developed a vaccine against dust-mite allergens that hasn't been used before," says Aliasger Salem, professor in pharmaceutical sciences at the UI and a corresponding author on the paper.

Dust mites are ubiquitous, microscopic buggers who burrow in mattresses, sofas, and other homey spots. They are found in 84 percent of households in the United States, according to a published, national survey. Preying on skin cells on the body, the mites trigger allergies and breathing difficulties among 45 percent of those who suffer from asthma, according to some studies. Prolonged exposure can cause lung damage.

Treatment is limited to getting temporary relief from inhalers or undergoing regular exposure to build up tolerance, which is long term and holds no guarantee of success.

"Our research explores a novel approach to treating mite allergy in which specially-encapsulated miniscule particles are administered with sequences of bacterial DNA that direct the immune system to suppress allergic immune responses," says Peter Thorne, public health professor at the UI and a contributing author on the paper. "This work suggests a way forward to alleviate mite-induced asthma in allergy sufferers."

The UI-developed vaccine takes advantage of the body's natural inclination to defend itself against foreign bodies. A key to the formula lies in the use of an adjuvant - which boosts the potency of the vaccine - called CpG. The booster has been used successfully in cancer vaccines but never had been tested as a vaccine for dust-mite allergies. Put broadly, CpG sets off a fire alarm within the body, springing immune cells into action. Those immune cells absorb the CpG and dispose of it.

This is important, because as the immune cells absorb CpG, they're also taking in the vaccine, which has been added to the package, much like your mother may have wrapped a bitter pill around something tasty to get you to swallow it. In another twist, combining the antigen (the vaccine) and CpG causes the body to change its immune response, producing antibodies that dampen the damaging health effects dust-mite allergens generally cause.

In lab tests, the CpG-antigen package, at 300 nanometers in size, was absorbed 90 percent of the time by immune cells, the UI-led team reports. The researchers followed up those experiments by giving the package to mice and exposing the

animals to dust-mite allergens every other day for nine days total. In analyses conducted at the UI College of Public Health, packages with CpG yielded greater production of the desirable antibodies, while lung inflammation was lower than particles that did not contain CpG, the researchers report.

"This is exactly what we were hoping for," says Salem, whose primary appointment is in the College of Pharmacy.

The researchers will continue to test the vaccine in the hope that it can eventually be used to treat patients.

The paper's first author is Vijaya Joshi, a graduate fellow in pharmacy at the UI. Contributing authors, all from the UI, include Andrea Dodd, Xuefang Jing, Amaraporn Wongrakpanich and Katherine Gibson-Corley.

The National Institutes of Health (grant numbers: P30 ES005605, R21 CA1 13345-01, R21 CA1 28414-01), the American Cancer Society and the UI's Lyle and Sharon Bighley professorship funded the research.

http://bit.ly/UxRc5v

Essays in English yield information about other languages

Grammatical habits in written English reveal linguistic features of non-native speakers' languages

Written by Larry Hardesty, MIT News Office

Computer scientists at MIT and Israel's Technion have discovered an unexpected source of information about the world's languages: the habits of native speakers of those languages when writing in English.

The work could enable computers chewing through relatively accessible documents to approximate data that might take trained linguists months in the field to collect. But that data could in turn lead to better computational tools.

"These [linguistic] features that our system is learning are of course, on one hand, of nice theoretical interest for linguists," says Boris Katz, a principal research scientist at MIT's Computer Science and Artificial Intelligence Laboratory and one of the leaders of the new work.

"But on the other, they're beginning to be used more and more often in applications. Everybody's very interested in building computational tools for world languages, but in order to build them, you need these features. So we may be able to do much more than just learn linguistic features. … These features could be extremely valuable for creating better parsers, better speech-recognizers, better natural-language translators, and so forth."

In fact, Katz explains, the researchers' theoretical discovery resulted from their work on a practical application: About a year ago, Katz proposed to one of his students, Yevgeni Berzak, that he try to write an algorithm that could automatically determine the native language of someone writing in English. The hope was to develop grammar-correcting software that could be tailored to a user's specific linguistic background.

Family resemblance

With help from Katz and from Roi Reichart, an engineering professor at the Technion who was a postdoc at MIT, Berzak built a system that combed through more than 1,000 English-language essays written by native speakers of 14 different languages.

First, it analyzed the parts of speech of the words in every sentence of every essay and the relationships between them. Then it looked for patterns in those relationships that correlated with the writers' native languages.

Like most machine-learning classification algorithms, Berzak's assigned probabilities to its inferences.

It might conclude, for instance, that a particular essay had a 51 percent chance of having been written by a native Russian speaker, a 33 percent chance of having been written by a native Polish speaker, and only a 16 percent chance of having been written by a native Japanese speaker.

In analyzing the results of their experiments, Berzak, Katz, and Reichart noticed a remarkable thing: The algorithm's probability estimates provided a quantitative measure of how closely related any two languages were; Russian speakers' syntactic patterns, for instance, were more similar to those of Polish speakers than to those of Japanese speakers.

When they used that measure to create a family tree of the 14 languages in their data set, it was almost identical to a family tree generated from data amassed by linguists. The nine languages that are in the Indo-European family, for instance, were clearly distinct from the five that aren't, and the Romance languages and the Slavic languages were more similar to each other than they were to the other Indo-European languages.

What's your type?

"The striking thing about this tree is that our system inferred it without having seen a single word in any of these languages," Berzak says. "We essentially get the similarity structure for free. Now we can take it one step further and use this tree to predict typological features of a language for which we have no linguistic knowledge."

By "typological features," Berzak means the types of syntactic patterns that linguists use to characterize languages - things like the typical order of subject, object, and verb; how negations are formed; or whether nouns take articles. A widely used online linguistic database called the World Atlas of Language Structures (WALS) identifies nearly 200 such features and includes data on more than 2,000 languages.

But, Berzak says, for some of those languages, WALS includes only a handful of typological features; the others just haven't been determined yet. Even widely studied European languages may have dozens of missing entries in the WALS database. At the time of his study, Berzak points out, only 14 percent of the entries in WALS had been filled in.

The new system could help fill in the gaps. In work presented last month at the Conference on Computational Natural Language Learning, Berzak, Katz, and Reichart ran a series of experiments that examined each of the 14 languages of the essays they'd analyzed, trying to predict its typological features from those of the other 13 languages, based solely on the similarity scores produced by the system. On average, those predictions were about 72 percent accurate.

Branching out

The 14 languages of the researchers' initial experiments were the ones for which an adequate number of essays - an average of 88 each - were publicly available. But Katz is confident that given enough training data, the system would perform just as well on other languages. Berzak points out that the African language Tswana, which has only five entries in WALS, nonetheless has 6 million speakers worldwide. It shouldn't be too difficult, Berzak argues, to track down more English-language essays by native Tswana speakers.

http://www.eurekalert.org/pub_releases/2014-07/vuot-btf072214.php

Boosting the force of empty space

Vacuum fluctuations may be among the most counter-intuitive phenomena of quantum physics.

Theorists from the Weizmann Institute and the Vienna University of Technology propose a way to amplify their force

Vacuum is not as empty as one might think. In fact, empty space is a bubbling soup of various virtual particles popping in and out of existence – a phenomenon called "vacuum fluctuations".

Usually, such extremely short-lived particles remain completely unnoticed, but in certain cases vacuum forces can have a measurable effect. A team of researchers from the Weizmann Institute of Science (Rehovot, Israel) and the Vienna University of Technology has now proposed a method of amplifying these forces by several orders of magnitude using a transmission line, channelling virtual photons.

"Borrowing" Energy, but just for a Little While

If you park your car somewhere and later it is gone, that is most probably not due to vacuum fluctuations. Objects do not disappear or reappear, that would violate the law of energy conservation. In the world of quantum physics, however, things are a bit more complicated. "Due to the uncertainty principle, virtual particles can come into existence for a brief period of time", says Igor Mazets from the Vienna University of Technology. "The higher their energy, the faster they will disappear again."

But such virtual particles can have a measurable collective effect. At very short distances, vacuum fluctuations can lead to an attractive force between atoms or molecules – the Van der Waals forces. Even the ability of a gecko to climb flat surfaces can in part be attributed to vacuum fluctuations and virtual particles.

The famous Casimir effect is another example of the power of the vacuum: The physicist Hendrik Casimir calculated in 1948 that two parallel mirrors in empty space will attract each other due to the way they influence the vacuum around them.

Atoms and Photons

Two atoms close to each other will also change the local vacuum around them. If one of them emits a virtual photon, which is almost instantly absorbed by the other, then on any timescale larger than the brief moment of the photon's existence, nothing much has happened – the total energy is conserved. But the fact that virtual particles can be exchanged modifies the vacuum around the atoms, and this leads to a force.

"Usually, such forces are very hard to measure", says Igor Mazets. "This is partly due to the fact, that such a photon may be emitted into any direction, and the chances of the second atom absorbing it are very small."

But what if the virtual particle has a little help to find its way? Ephraim Shahmoon, Gershon Kurizki (Weizmann Institute of Science) and Igor Mazets calculated what happens to vacuum forces between atoms when they are placed in the vicinity of an electrical transmission line such as a coaxial cable or a coplanar waveguide (a device used in cavity quantum electrodynamics experiments as an open transmission line), cooled to very low temperatures.

"In that case, the fluctuations are effectively confined to one dimension", says Igor Mazets. The virtual particles will be forced to go into the direction of the other atom.

In that case, the fluctuation-mediated attraction between the atoms becomes orders of magnitude stronger than in free space. Usually, the force decreases rapidly with increasing distance between the atoms. Due to the transmission line, it falls off with one over the distance cubed, instead of one over the seventh power of the distance, as in the usual case.

The researchers believe that their proposed enhancement of the power of vacuum fluctuations can have profound implications for understanding Casimir- and Van der Waals forces and it may even be used for applications in quantum information processing and other emerging quantum technologies.

http://bit.ly/1mN4F0e

Transistor Successor Set to Bring on "The Machine" Age Soon

A successor to an essential part in today's computers may arrive in just a few years

Jul 22, 2014 |By Wendy M. Grossman

A replacement for the ordinary transistor may make it to market by the end of this decade, an event that will herald a radical redesign of traditional computer architectures. The memristor, the subject of much study over the last six years, could become the basic building block for an array of new devices - from the sensors and memory chips being built into the "Internet of Things" (connected, sensor-embedded devices) to the giant computers used for big data applications by scientists, engineers and Wall Street.

Today, and for the past 50 years, computers have worked by processing data in fast dynamic memory and pushing it down wires - input/output channels - to slower-speed permanent disk storage. Memristors may combine into a single device the best characteristics of both dynamic memory (the RAM in a desktop computer) and hard drives or flash memory chips, which retain data when the electricity goes off.

The original idea dates back to the late 1990s, when Senior HP Fellow Stan Williams set up Hewlett–Packard's Information and Quantum Systems Laboratory to scope out the next two decades of computing. For 40 years the industry has relied on its ability to manufacture ever-shrinking, ever-cheaper transistors based on Moore's Law (the observation made by Intel founder Gordon Moore in 1965 that the number of transistors that can fit on a chip doubles about every two years). Williams' team accordingly began by studying increasingly small transistors, which led them to consider what would happen when the devices shrink to the size of individual molecules, in which the movement of a single atom would affect performance. At that size, the researchers encountered an effect they didn't understand until 2008, when one of the team read a paper written more than 35 years earlier by Leon Chua, a professor in electrical engineering and computer sciences at University of California, Berkeley.

In it Chua calculated that memristors would become a fourth electronic component, along with resistors, capacitors and inductors. Williams recognized that his team was seeing Chua's prediction materialize in a thin film of titanium dioxide. Subsequently, others have joined the search. In 2012 HRL Labs, a research facility jointly owned by General Motors and Boeing, announced the first successfully functioning memristor array - built with the complementary metal-oxide semiconductor (CMOS) manufacturing process used for most electronic devices.

The old and the new electronics function in fundamentally different ways. Transistors toggle between an on or off state, whereas memristors, like analog devices, can occupy a range of in-between states. Developers had expected memristor development to proceed more quickly than it has. In 2010 HP predicted that memristor devices would reach the market this year. Not likely, according to Kirk Bresniker, HP Labs chief architect and HP fellow. The devices still need more work before they are ready for commercial release. HP and the company's development partners are still scouring the periodic table looking for the precise combination of elements and the specific manufacturing processes that will allow the best memristive effect to preserve data intact. They also want to incorporate this technology into standard CMOS chips that can be mass-manufactured at a reasonable cost.

Meanwhile the concept of what can be built with memristors has continued to evolve. At HP's Discover Conference in mid-June, company chief technology officer Martin Fink outlined a simple architecture he called simply "The Machine." It consists of a set of memory circuits connected using optical fibers instead of copper wires to connect to highly efficient special-purpose processors.

The industry has several goals in making the shift. Memristors can vastly improve energy efficiency of electronic components, and are better able to cope with the floods of data expected from the Internet of Things, which monitor or control equipment or systems in factories, office buildings or homes. Essential to their development is a continuation of the exponential growth in computing power and storage density that has seen prices plunge over the past 40 years. For similar reasons, IBM has just announced it will spend $3 billion to pursue experimental "post-silicon" architectures and chips, predicting a fundamental revamping of existing systems in 10 years.

These changes will produce a fundamental overhaul of computer operating systems to accommodate hardware that no longer differentiates between dynamic memory and long-term storage. Bresniker sees the change as an opportunity to jettison layers of cumbersome operating system code that was previously adopted to accommodate the limitations of older hardware.

HP's current development timetable has memristors going into the earliest stage of production in 2015 and launching as DIMMs (dual in-line memory modules) for computer memory in 2016. The operating system for "The Machine" will go into wider public beta testing in 2017, and the new architecture is intended to be integrated into actual products in 2019. Even if none of this pans out, Bresniker believes the attempt is worth it: "Each of the elements is interesting…[on its own]. Pulling out that copper and dropping in that piece of fiber will be more efficient, even with a traditional computing and memory regime all around it…. We need a

replacement memory technology. If it does nothing else than drop in where my DIMMs drop in today, that will be a useful thing."

http://www.eurekalert.org/pub_releases/2014-07/uoth-aas072214.php

Anti-pain agent shrinks oral cancers, leaves healthy tissues alone

Mouse models of human oral cancer treated with an agent called capsazepine showed dramatic tumor shrinkage without damage to surrounding tissues

SAN ANTONIO - Mouse models of human oral cancer treated with an agent called capsazepine showed dramatic tumor shrinkage without damage to surrounding tissues, researchers from the School of Dentistry and School of Medicine at The University of Texas Health Science Center at San Antonio found. The Health Science Center has claimed intellectual property on results of the study, which is described in the journal Oral Oncology.

Late diagnosis, low survival

Oral squamous cell carcinoma is the eighth most common cancer in the U.S. with 40,000 new cases and nearly 8,000 deaths reported annually. "These tumors develop primarily on the side of the tongue," said study first author Cara B. Gonzales, D.D.S., Ph.D., assistant professor of comprehensive dentistry and an investigator with the Cancer Therapy & Research Center at the UT Health Science Center at San Antonio. "Unfortunately, 60 percent of patients have large tumors before seeking help, and their five-year survival rate is as low as 30 percent."

Pain blocker and other properties

Capsazepine was developed to block TRPV1, a calcium channel found in pain-sensing neurons. When TRPV1 is activated, a "pain signal" is sent to the brain. Capsazepine may reduce oral cancer pain because it blocks tumor-secreted factors from stimulating TRPV1 on these neurons. Dr. Gonzales found that capsazepine also has anti-cancer activity that may be associated with its ability to increase oxidative damage in tumors. Enhanced oxidative stress leads to auto-destruction of tumor cells, the researchers theorize.

"Here's the beauty," Dr. Gonzales said. "Capsazepine kills cancers selectively, leaving normal tissues alone, and also acts on neurons to block pain, a desirable combination in a potential medication."

Hope for future: systemic administration

So far, only local administration of capsazepine, directly into the primary tumors, has been tested. But many patients with oral cancer have disease that has spread. "We would like to be able to deliver this therapy systemically to target metastatic disease," Dr. Gonzales said. "Our laboratory is working with the Center for Innovation in Drug Discovery, a partnership between the Health Science Center and UTSA, to develop novel drugs that are similar to capsazepine with improved efficacy for the purpose of systemic administration to treat tumors that are inaccessible to local injection or that have metastasized."

At-risk patients

Randal A. Otto, M.D., F.A.C.S., professor and chairman of the Department of Otolaryngology-Head & Neck Surgery in the School of Medicine, said: "These tumors, if identified and treated early, are definitely curable. Unfortunately, most patients present with advanced disease with the cancer involving critical structures. This markedly decreases the chance for cure and dramatically increases the risks associated with treatment. Anything that selectively attacks the tumor while not injuring the normal tissues can only help the patient."

Acknowledgments

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Vanilloids induce oral cancer apoptosis independent of TRPV1

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When will we take medicinal honey seriously?

Honey is now regularly being shown to kill superbugs in the laboratory and save patient's limbs on hospital wards, but why is its medicinal use still so limited in the UK?

By Zoe Gough Reporter, BBC Nature

The antibacterial properties of honey have long been known, both ancient Greek and Egyptian physicians are said to have valued it and it was used in the treatment of wounds right up to World War Two.

Honey's reputation was relegated to that of an old wives' tale in the twentieth century after the discovery of penicillin heralded the widespread use of antibiotic drugs to combat infections.

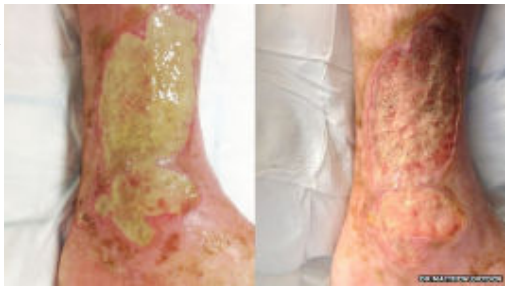
But with antibiotic resistance now high on the global agenda, scientists and doctors are working together to once more prove honey's effectiveness in battling life-threatening bacteria.

Researchers say honey has been successful in treating severe wounds including ulcers, pressure sores, trauma injuries and infected surgical wounds - reducing the reliance on antibiotics and providing an alternative to antiseptics which can harm healing tissue.

Filtered or medical-grade honey is used in licensed wound-care products around the world.

However large-scale randomised clinical trials have yet to take place in this country so its use remains low compared with other wound treatments like silver and iodine.

Those looking into its curative potential claim this may be due to it being a natural product which attracts scepticism from medical scientists. Organisations which fund medical research say no such stigma exists, grant applications simply need to be robust.



Engineered honey successfully treated a patient who was facing amputation due to large ischaemic ulcers which had become infected with pseudomonas bacteria.

Sam Edwards, a maintenance engineer from Wrexham, Wales, is a recent convert to the power of honey, after developing a rare skin condition caused by a cut from a koi carp infected with Mycobacterium marinum.

"The pain is like having a bath in a deep fat fryer 24 hours a day," Sam said. "It put me in a wheelchair for a long time as well as meaning long stays in hospital and mechanical dermabrasion."

Antibiotics caused jaundice and doctors began to talk about multiple amputations so Sam looked around for alternative treatments and tried everything from steroids to maggots but nothing worked.

In December 2012 Sam was introduced to manuka honey dressings by a street doctor from Venezuela. By January 2014 Sam had found a UK supplier and began the treatment. "It has turned my life around. It hurts a little bit the first time you use it but in the space of five months I am almost completely healed, it's amazing," Sam said. Manuka honey comes from the New Zealand manuka plant and has been available on prescription in the UK for the last 10 years.

Professor Rose Cooper, from the Centre for Biomedical Sciences at Cardiff Metropolitan University, has been at the forefront of its research since the late 1990s.

Using electron microscopy, which can reveal the structure of bacteria, she has shown even low concentrations of the honey stops bacteria including MRSA growing, meaning cells cannot divide and therefore are unable to form infections.

Combining honey with oxacillin and other antibiotics has also been shown to be more effective against antibiotic resistant bacteria. She is currently investigating how different bacteria seem to be affected in different ways by manuka honey, believing it to have a wider application than just killing bugs.

Prof Cooper said she has often found it difficult to get her research published but admits the scientific standards of clinical work with honey has been varied.

"One of the problems is a good clinical trial should be a double blind so that neither the patient nor the practitioner will know which of the patients are having the intervention that's being tested," she said.

"The trouble with honey is the patients know its sticky, they can smell it, and of course the practitioners know too, so it's very difficult to achieve that best quality."

In a clinical setting, research has so far been small-scale, but dramatic results have been reported. Dr Matthew Dryden, consultant in infection and microbiology at Hampshire Hospitals NHS Foundation Trust, has seen a number of patients' wounds transformed by honey.

The smart skills behind honey

He uses an engineered version, called Surgihoney, as a wound dressing after carrying out laboratory tests against bacteria gathered from infected wounds.

Surgihoney killed all of the bugs including multiple drug-resistant ones like MRSA, Ecoli and pseudomonas aeruginos and its effects were comparable to commonly-used antiseptics, which can have adverse side effects.

"There was one man with an ischaemic leg, where it was really a choice between amputating the leg and/or giving him potent systemic antibiotics," Dr Dryden told BBC Nature.

"The ulcer was heavily colonised with Pseudomonas aeruginosa, which is a nasty, often resistant bug so we put daily (Surgihoney) dressings on the ulcer. "By day eight the bacteria had completely disappeared and the ulcer had started to get better, so for the time being it had saved his leg, it prevented him from having antibiotics and got him out of hospital."

Dr Dryden has also shown that the product can reduce infection rates following caesarean sections and also those associated with cancer patients receiving chemotherapy treatment through intravenous lines. Like Prof Cooper, Dr Dryden also believes honey products have more potential uses and would like to carry out full randomised trials. But an application for funding to carry out such research was rejected by the National Institute for Health Research (NIHR).

"Despite all the publicity about antibiotic resistance, using a so-called natural product is not terribly sexy, scientifically," Dr Dryden said. "There's a lot of snake oil salesmen out there who claim all sorts of things for all sorts of natural products. "But we've actually got more patient data than many wound dressing treatments. I think because its honey it seems a bit alternative and that puts the scientists off."

High quality trials could also be carried out by big wound care companies. The father and son team behind Surgihoney are talking to several firms about developing it commercially.

The idea began on a farm in Chile owned by former managing director Ian Staples. Having decided to keep bees on the farm he spotted that the honey they produced did not spoil in the hive, suggesting natural antimicrobial activity. In fact, an enzyme inside honey produces hydrogen peroxide which is a well-known disinfectant.

Ian and his son, Stuart, who now live in West Sussex, commissioned scientists to develop a product which boosted the antibacterial properties of any organic honey. "When we first started this the doctors we worked with said this was as big a breakthrough as penicillin," Stuart Staples said. "Whether it is or not that's why we bet the farm on it."

New funding

He estimates that health organisations in the UK currently spend less than £3 million ($5.1 million USD) a year on honey dressings, compared with £16 million ($27.2 million USD) spent annually by the NHS on antiseptic silver products.

"Dr Margaret Chan from the World Health Organisation said in the future a scratched knee could kill you and we're saying 'no it couldn't' because we have a device that no bug can survive contact with," Mr Staples said.

Organisations which fund medical research in the UK say all grant applications are subject to peer review and judged in open competition. A Department of Health spokesperson said: "Britain's reputation as a world leader in science, research and development depends upon innovative approaches to improving treatments and finding new cures.

"The NIHR welcomes funding applications for research into any aspect of human health, including in this case, the use of honey as an antimicrobial agent."

The Wellcome Trust said it has not funded any research into honey but said this could be for a variety of reasons including no bids being made, bids not being relevant to the funding criteria or the science not being of high enough quality.

The Medical Research Council (MRC) said it had also not funded any studies with honey but that did not mean it would not do so in the future.

It added that it is currently inviting bids for funding as part of a new collaboration between all seven UK research councils to tackle antimicrobial resistance.

http://www.bbc.com/news/science-environment-28442640

Deep sea mining licences issued

Vast new areas of the ocean floor have been opened up in an accelerating search for valuable minerals including manganese, copper and gold.

In a move that brings closer a new era of deep sea mining, the UN's International Seabed Authority (ISA) has issued seven new exploration licences.

State-owned and private companies from India, Brazil, Singapore and Russia are among those to land permission for minerals prospecting.

One British firm, UK Seabed Resources, a subsidiary of the US defence giant Lockheed Martin, has secured exploration rights to an area larger than the entire UK.

This means that the total area of seabed now licensed in this new gold rush has reached an immense 1.2 million square kilometres under 26 different permits for minerals prospecting.

Deep sea mining is a new frontier in the quest for the precious raw materials needed for modern economies but environmental groups have long warned of the potential damage to marine ecosystems.

Mining the ocean floor was first investigated in the 1960s but only recently have technological advances - spurred by the oil and gas industry - and high prices for resources combined to make operations feasible.

The ISA was set up to manage the exploitation of the ocean floor beyond territorial limits to prevent a free-for-all and has so far only issued licences for exploration. The first permits for exploitation could come in the next few years.

Michael Lodge of the ISA told the BBC: "There's definitely growing interest. Most of the latest group are commercial companies so they're looking forward to exploitation in a reasonably short time - this move brings that closer."

Still to be negotiated are the conditions and rules for actual mining.

A protocol to minimise the environmental impact is still being drawn up.

And arrangements for royalties to be paid to developing and landlocked countries have yet to be settled - a basic principle of the ISA is that seabed riches should be shared globally.

Two of the new licences - for German and Indian organisations - cover deep ocean ridges where hydrothermal vents have created potentially rich deposits.

Dr Jon Copley of the University of Southampton, a marine biologist, has monitored the development of deep sea mining amid concerns about its possible effects on the natural world.

"In total, about 6,000 km of mid-ocean ridge in international waters are now being explored for potential seafloor mining.

In total, around 7.5% of the global mid-ocean ridge - the geological backbone of our planet - is now being explored for its mineral wealth. "Ridges are one of the three deep-sea environments where there are mineral deposits attracting interest, in this case for the metal ores that form at deep-sea vents along the ridges. "But those vents are also home to colonies of some species that aren't found in other deep ocean environments, which may make them susceptible to environmental impacts from mining."

UK Seabed Resources (UKSRL) conducted a baseline environmental survey of its licence area in the Pacific last October. It is hoping to extract so-called nodules from the ocean floor - small lumps of rock which contain far higher proportions of metals than ores found on land.

Duncan Cunningham of UKSRL said the company remained "committed to environmentally responsible, transparent and commercially sound development of the area". He added: "We were extremely pleased to have had the opportunity to present details of our first environmental baseline cruise to the ISA and other stakeholders."

The first seabed mine is likely to be in the waters off Papua New Guinea. In a deal arranged outside the ISA system, a Canadian company, Nautilus Minerals, plans to extract metals from a field of hydrothermal vents.

The project was delayed for years by a dispute with the PNG government but terms have now been finalised and huge robotic mining machines are being constructed.

http://nyti.ms/1l2BKWg

A Dearth in Innovation for Key Drugs

There is clearly something wrong with pharmaceutical innovation.

Antibiotic-resistant infections sicken more than two million Americans every year and kill at least 23,000.

The World Health Organization has warned that a "post-antibiotic era" may be upon us, when "common infections and minor injuries can kill."

Even the world's tycoons consider the proliferation of antibiotic-resistant bacteria one of the crucial global risks of our times, according to a survey by the World Economic Forum.

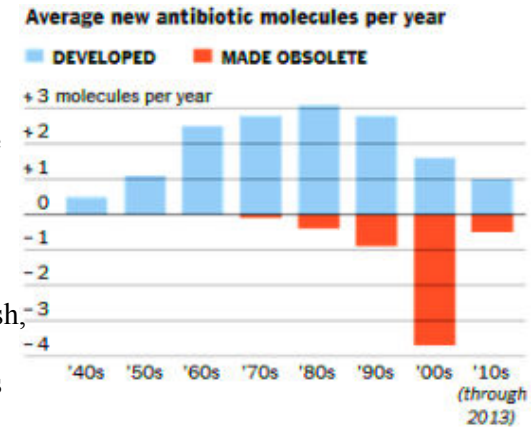
Yet the enthusiasm of the pharmaceutical industry for developing drugs to combat No major new type of antibiotic has been developed since the late 1980s, according to the W.H.O. From 2011 to 2013, the Food and Drug Administration approved only three new molecular entities to combat bacterial diseases - the lowest rate since the 1940s.

"No sane company will develop the next antibiotic," said Michael S. Kinch, who led a team at the Yale Center for Molecular Discovery tracking the evolution of pharmaceutical innovation over the last two centuries.

More Bugs, Fewer Drugs

And this is hardly the drug industry's only problem. Antibiotics, Professor Kinch told me, "are the canary in the coal mine."

This is particularly striking at a time when the pharmaceutical industry is unusually optimistic about the future of medical innovation. Dr. Mikael Dolsten, who oversees worldwide research and development at Pfizer, points out that if progress in the 15 years until 2010 or so looked sluggish, it was just because it takes time to figure out how to turn breakthroughs like the map of the human genome into new drugs.



The pace of development of new antibiotic molecules has slowed sharply since its peak in the 1980s, even as drug-resistant bacteria and other factors have made old antibiotics obsolete. **Source: Michael S. Kinch, Denton Hoyer, et. al., Yale Center for Molecular Discovery.** ***This is a link to the paper.***

The pipeline today, which includes tailored treatments for cancer, newfangled vaccines and therapies for tough diseases like hepatitis C, is robust.

So far this decade, the F.D.A. has approved drugs at a pace second only to the 1990s. In 2012, the FDA approved 37 new drugs, the most in 15 years.

But the economics of the drug development, argues Professor Kinch, who in July was appointed associate vice chancellor of Washington University in St. Louis, are not conducive to creating the highest levels of public health.

More and more antibiotics are going out of circulation every year - either because of bacteria have become resistant to them or because they have been replaced by better or less toxic drugs. The pharmaceutical arsenal against bacterial infections shrank to only 96 different molecules by the end of last year, 17 fewer than at the turn of the century.

Nevertheless, many of the big drug companies that produced the antibiotic breakthroughs of the past have decided to drop this line of research. And few new entrants are jumping in.

"It has become very difficult to find new drug classes to fight infections," Dr. Dolsten of Pfizer acknowledged. "There haven't been enough incentives for the industry to take on 10 or 15 years of research."

Antibiotics face a daunting proposition. They are not only becoming more difficult to develop, but they are also not obviously profitable. Unlike, say, cancer drugs, which can be spectacularly expensive and may need to be taken for life, antibiotics do not command top dollar from hospitals. What's more, they tend to be prescribed for only short periods of time.

Importantly, any new breakthrough antibiotic is likely to be jealously guarded by doctors and health officials for as long as possible, and used only as a drug of last resort to prevent bacteria from developing resistance. By the time it became a mass-market drug, companies fear, it could be already off patent and subject to competition from generics that would drive its price down.

Antibiotics are not the only drugs getting the cold shoulder, however. Research on treatments to combat H.I.V./AIDS is also drying up, according to the research at Yale, mostly because the cost and time required for development are increasing.

Research into new cardiovascular therapies has mostly stuck to less risky "me too" drugs.

Neuropsychiatric diseases, including Alzheimer's and depression, are the leading cause of disability across most of the industrial world. And they are going to get worse. Yet researchers have underscored a dearth of investment into these diseases.

Instead, pharmaceutical and biotechnology firms are betting on personalized therapies - mostly targeting specific varieties of cancers - and drugs for so-called orphan diseases, which affect very small populations.

"More people are studying orphan diseases than have orphan diseases," Professor Kinch said jokingly. Of the new drugs that the F.D.A. approved in 2013, about 70 percent were specialty drugs - which are used by less than 1 percent of the population, according to the drug benefits manager Express Scripts.

The problem, of course, lies in the industry's incentives. The cost of developing a new drug has skyrocketed over the last three decades. A research paper by scientists from Eli Lilly suggested that in 2010, it cost $1.8 billion to bring a big new drug from conception to rollout, through the costly gantlet of clinical trials needed to prove that it is both safe and more effective than existing therapies.

Developing orphan drugs is cheaper.

They receive expedited approval from the F.D.A. Clinical trials are inherently less expensive because the drugs are aimed at a small population. And insurance companies are willing to pay $100,000 a year for a drug that few patients will use.

"Companies are flocking to rare diseases," said John LaMattina, a former head of research at Pfizer who now writes a blog about pharmaceutical research. "They might only make $500 million in sales a year, but their costs are much lower."

Similar considerations have pushed pharmaceutical companies into newfangled biological drugs at the expense of old-fashioned compounds.

Standard brand-name drugs lose 80 percent of the market within a year of patent expiration. Biologicals face much less generic competition, protected both by regulation and the fact that it is tough to determine the equivalency of different biological agents.

The wave of protests over the $84,000 cost per course of Gilead's blockbuster new drug to treat hepatitis C, Sovaldi, highlights the kind of strain that can be caused when mass market therapies are priced like niche specialty drugs.

"I've seen nothing as potentially harmful as the exorbitant pricing displayed by Gilead," wrote Dr. Steve Miller, the chief medical officer of Express Scripts. Regardless of whether that is worth it for the individual patient or society at large - which it probably is - the price could bankrupt Medicaid budgets around the country.

Can drug makers' incentives be fixed? Some argue that the patent system governing drug innovation is not up to the task, and suggest handing over most drug research and development to the National Institutes of Health, which already spend tens of billions on basic research.

Tweaking the existing system might be a more feasible proposition, however. Research on new antibiotics could be encouraged by allowing shorter clinical trials for the promising molecules or guaranteeing minimum returns for groundbreaking drugs.

Patricia Danzon of the Wharton School of the University of Pennsylvania suggests recalibrating the regulatory burden to favor research in drugs with a broader potential footprint. "The decks have been stacked in favor of orphan drugs," she said.

At the same time, new mechanisms are needed to constrain prices.

The National Health Service in Britain may have a bad reputation in the United States, but Americans could benefit from something like the country's National Institute for Health and Care Excellence, which determines what therapies will be covered, based on their efficacy and their price.

"There's a myth in the United States that market forces are working to control prices," Professor Danzon said. It's clear that they aren't. But the market isn't delivering the innovation we need, either.

http://nyti.ms/1qENGFq

Inside Man's Best Friend, Study Says, May Lurk a Green-Eyed Monster

Any dog owner would testify that dogs are just as prone to jealousy as humans. But can one really compare Othello's agony to Roscoe's pique?

By JAMES GORMAN JULY 23, 2014

The answer, according to Christine Harris, a psychologist at the University of California, San Diego, is that if you are petting another dog, Roscoe is going to show something that Dr. Harris thinks is a form of jealousy, even if not as complex and twisted as the adult human form.

Other scientists agree there is something going on, but not all are convinced it is jealousy.

And Roscoe and the rest of his tribe were, without exception, unavailable for comment.

Dr. Harris had been studying human jealousy for years when she took this question on, inspired partly by the antics of her parents' Border collies.

When she petted them, "one would take his head and knock the other's head away," she said. It certainly looked like jealousy.

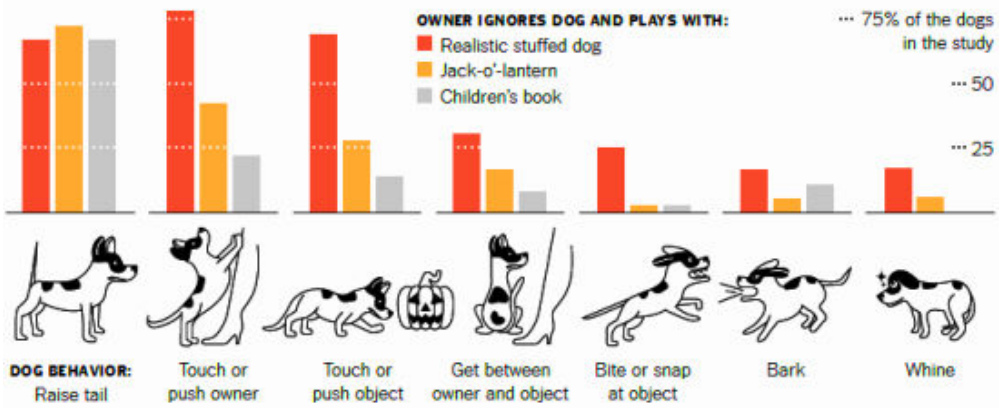
But having studied humans, she was aware of different schools of thought about jealousy.

Some scientists argue that jealousy requires complex thinking about self and others, which seems beyond dogs' abilities.

Others think that although our descriptions of jealousy are complex, the emotion itself may not be that complex.

Your Jealous Dog

In a study, dogs reacted more strongly when their owners paid attention to stuffed dogs than to more generic objects.



By The New York Times; Illustrations by Jennifer Daniel Source: PLoS One

Dog emotions, as owners perceive them, have been studied before. In one case, Alexandra Horowitz, a cognitive scientist who is an adjunct associate professor at Barnard College and the author of "Inside of a Dog," found that the so-called guilty look that dogs exhibit seemed to be more related to fear of punishment.

Dr. Harris ventured into the tricky turf of dog emotion by devising a test based on work done with infants.

When dog owners petted and talked to a realistic stuffed dog that barked and whined, the people's own dogs came over, pushed the person or the stuffed dog, and sometimes barked.

After the experiment, many of the dogs sniffed the rear end of the stuffed dog, suggesting, Dr. Harris said, that the dogs thought it might be real.

Dr. Harris also recorded what happened as the owners petted and talked to a jack-o'-lantern and read a children's book aloud, to see if any old distraction would provoke a reaction.

The dogs paid little attention to the jack-o'-lantern and very little to the book.

Dr. Harris concluded, in a paper in PLoS One written with Caroline Prouvost, also at the University of California, San Diego, that the dogs showed a "primordial" form of jealousy, not as complex as the human emotion, but similar in that there is a social triangle and the dog is trying to make sure it, not the rival, receives the attention.

"What can be shown is that dogs seem to want an owner's attention when there is attention being given out," she said. "This study confirms that."

Sybil Hart, at Texas Tech, who has studied jealousy in infants, said she thought the research was "very well done and makes a very compelling argument."

If one sees jealousy in babies and dogs, she said, "to some degree, it's innate," which would be important to know for attempts to manage human jealousy.

"Overall, trying to make it go away has not been very successful," Dr. Hart said. "We are trying to eliminate jealousy, and scientists are saying maybe we should try to understand it better."

Jealousy, Dr. Harris wrote in the study, is "the third leading cause of nonaccidental homicide across cultures."

Whatever the dogs' behavior is called, said Brian Hare, a director of the Duke Canine Cognition Center at Duke University, there are practical implications for their owners.

"Attention seeking can lead to jealousylike behavior in dogs that includes aggression in some cases," he said.

"So for dogs with suspected aggression problems, it may be important to avoid situations where they feel ignored."

http://www.eurekalert.org/pub_releases/2014-07/cru-nir072214.php

No increased risk of cancer near Sellafield or Dounreay in recent years

Children, teenagers and young adults living near Sellafield or Dounreay since the 1990s are not at an increased risk of developing cancer according to research published in the British Journal of Cancer today* (Wednesday).

Researchers from the Childhood Cancer Research Group at the University of Oxford and from Newcastle University studied cancer rates between 1963 and 2006 among those who were under 25 and living near Sellafield or Dounreay when diagnosed. No difference was found in cancer incidence from 1991-2006 between those living near these nuclear power plants and the general population. But the study confirmed the raised risks of cancer, particularly leukaemia, already reported for earlier time periods.

Kathryn Bunch, lead author on the study, said: "For many years, there have been concerns over the potential raised cancer risk among people – particularly children – who live near nuclear installations. This study found that children, teenagers and young adults living close to Sellafield and Dounreay are no longer at an increased risk of developing cancer.

"Furthermore, there is no evidence of any increased risk of cancer later in life for those who were born near these power plants."

The numbers of cancers observed around Sellafield and Dounreay were compared with those expected from national cancer registration rates using census derived population estimates.

Overall, leukaemia is the eleventh most common cancer in the UK, but it accounts for around a third of all cancers diagnosed in children.

Dr Julie Sharp, Cancer Research UK's head of health information, said: "There has been a lot of concern that nuclear power stations could increase the risk of cancer, particularly leukaemia. This study is reassuring for anyone who happens to be living near a power plant, as it shows no increased risk among children, teenagers or young adults in recent years."

http://www.eurekalert.org/pub_releases/2014-07/uoc--gis072314.php

Gene inhibitor, salmon fibrin restore function lost in spinal cord injury

UCI Reeve-Irvine researchers identify novel combination treatment

Irvine, Calif - A therapy combining salmon fibrin injections into the spinal cord and injections of a gene inhibitor into the brain restored voluntary motor function impaired by spinal cord injury, scientists at UC Irvine's Reeve-Irvine Research Center have found.

In a study on rodents, Gail Lewandowski and Oswald Steward achieved this breakthrough by turning back the developmental clock in a molecular pathway critical to the formation of corticospinal tract nerve connections and providing a scaffold so that neuronal axons at the injury site could grow and link up again. Results appear in the July 23 issue of The Journal of Neuroscience.

The work expands on previous research at UCI. In 2010, Steward helped discover that axons flourish after the deletion of an enzyme called PTEN, which controls a molecular pathway regulating cell growth. PTEN activity is low during early development, allowing cell proliferation. PTEN subsequently turns on, inhibiting this pathway and precluding any ability to regenerate.

Two years later, a UCI team found that salmon fibrin injected into rats with spinal cord injury filled cavities at the injury site, giving axons a framework in which to reconnect and facilitate recovery. Fibrin is a stringy, insoluble protein produced by the blood clotting process and is used as a surgical glue.

"This is a major next step in our effort to identify treatments that restore functional losses suffered by those with spinal cord injury," said Steward, professor of anatomy & neurobiology and director of the Reeve-Irvine Research Center, of the current findings. "Paralysis and loss of function from spinal cord injury has been considered irreversible, but our discovery points the way toward a potential therapy to induce regeneration of nerve connections."

In their study, he and Lewandowski treated rodents with impaired hand movement due to spinal cord injury with a combination of salmon fibrin and a PTEN inhibitor called AAVshPTEN. A separate group of rodents got only AAVshPTEN. The researchers saw that rats receiving the inhibitor alone did not exhibit improved motor function, whereas those given AAVshPTEN and salmon fibrin recovered forelimb use involving reaching and grasping.

"The data suggest that the combination of PTEN deletion and salmon fibrin injection into the lesion can significantly enhance motor skills by enabling regenerative growth of corticospinal tract axons," Steward said.

According to the Christopher & Dana Reeve Foundation, about 2 percent of Americans have some form of paralysis resulting from spinal cord injury, due primarily to the interruption of connections between the brain and spinal cord. An injury the size of a grape can lead to complete loss of function below the site of occurrence. For example, an injury to the neck can cause paralysis of the arms and legs, an absence of sensation below the shoulders, bladder and bowel incontinence, sexual dysfunction, and secondary health risks such as susceptibility to urinary tract infections, pressure sores and blood clots due to an inability to move the legs.

Steward said the next objective is to learn how long after injury the combination treatment can be effectively administered. "It would be a huge step if it could be delivered in the chronic period weeks and months after an injury, but we need to determine this before we can engage in clinical trials," he said.

Lewandowski is a project scientist in the Reeve-Irvine Research Center. The study received support from the National Institutes of Health (grant R01 NS047718) and donations from Cure Medical and Unite 2 Fight Paralysis.

http://www.eurekalert.org/pub_releases/2014-07/acoe-kcb072314.php

Ketamine can be a wonder drug for ER patients and their physicians

Ketamine can safely provide analgesia, sedation and amnesia for rapid, life-saving intubation for critically ill patients arriving at the emergency department

WASHINGTON - For critically ill patients arriving at the emergency department, the drug ketamine can safely provide analgesia, sedation and amnesia for rapid, life-saving intubation, despite decades-old studies that suggested it raised intracranial pressure. The results of a systematic review of 10 recent studies of what many emergency physicians regard as a "wonder drug" are published online in Annals of Emergency Medicine "The Effect of Ketamine on Intracranial and Cerebral Perfusion Pressure and Health Outcomes: A Systematic Review."

"Apprehension for many years about ketamine's effects on blood pressure or injured brains inhibited its use for intubation, especially in North America compared to Europe, but our review shows those concerns are likely overblown," said lead study author Corinne Hohl, MD, of the Department of Emergency Medicine at Vancouver General Hospital in Vancouver, Canada. "In view of recent concerns about the potential negative effects of an alternative induction agent, etomidate, ketamine should be considered routinely in patients with life-threatening infections and more regularly for patients who have been 'found down,' or unconscious, before being transported to the ER."

The most significant worry about ketamine in critically ill patients has been its effect on intracranial and cerebral pressures. Studies comparing ketamine to sufentanil, fentanyl and other pharmacological agents (vasopressors, neuromuscular blocking agents, sedatives) found no differences in intracranial and cerebral pressures of patients who had been treated with them.

Studies assessing patients sedated with ketamine found no difference in neurological outcomes compared to patients sedated with fentanyl, sufentanil, remifentanil or etomidate. Length of stay in the hospital was unaffected by ketamine use. Patients sedated and intubated with ketamine were also no likelier to die than patients sedated by other agents.

"Given the potential benefit to emergency patients and their physicians, the debate on ketamine should be settled by a large, randomized controlled trial," said Dr. Hohl. "In the meantime, our review suggests what many emergency physicians already believe is true: Ketamine is safe and incredibly useful in critically ill patients who require rapid intubation."

http://bit.ly/WU1U8e

Oxygen oasis for early life found in ancient rock

First hard evidence of an oxygen oasis preserved in ancient rocks

23 July 2014 by Michael Marshall

FOR the first half of Earth's existence, there was no oxygen to be had. The air wasn't breathable and life in the oceans was little more than primitive sludge. Even in this hostile world, though, small "oases" of oxygen-rich water persisted, fuelled by bacteria. Now it seems we have found the first hard evidence of one of these oxygen oases, preserved in ancient rocks.

If a person were to step out into this ancient world, they would die of asphyxiation within minutes. It wasn't until around 2.4 billion years ago that oxygen flooded the oceans and gave rise to the air and seas we recognise.

"But many researchers have suspected that the first biological production of oxygen began long before that," says Timothy Lyons of the University of California, Riverside. Rocks from 4 to 2.5 billion years ago often contain bands of iron-rich minerals. These formed when bacteria started pumping out oxygen, which reacted with dissolved iron in the ocean to form particles of rock that sank to the bottom.

So oxygen might have built up in isolated pockets – perhaps in shallow seas cut off from the global ocean. "The idea of oxygen oases in ancient seas has been around for a long time, but no one was able to pinpoint a specific example of such an oasis," says Robert Riding of the University of Tennessee in Knoxville. He and his colleagues now say they have found one.

They collected rock samples from Steep Rock Lake in Ontario, Canada. Rocks there are 2.8 billion years old and contain a mixture of iron minerals and limestone, as well as the remains of thin mats of microbes called stromatolites. The area was once "a shallow shelf, partly isolated from the open sea by a stromatolite reef, and close to a land mass that could have supplied nutrients", says Riding.

"Steep Rock is one of the oldest thick limestones on Earth, and is certainly the best preserved," says Riding. His team's analyses of the limestone suggest it has not changed since it was laid down.

That is key, adds Riding, because the calcium carbonate that makes up limestone can only form in water that has first been stripped of all its dissolved iron. He says

the best explanation for the presence of the mineral is that bacteria pumped out oxygen, which reacted with all the iron in the water (Precambrian Research, doi.org/tsq).

The oasis only persisted for about 5 million years, though. After that, sea levels rose, overtopped the reef and swamped the area with a fresh influx of iron, causing oxygen levels to crash. "The existence of the oases was tenuous," says Lee Kump of Penn State University in University Park.

Despite their name, the oases were dangerous places. Because oxygen is chemically reactive, when it first built up it was a deadly pollutant. Bacteria living in the oases would have been forced to evolve oxygen-defence mechanisms, or die. So the oases would have pushed early life to adapt to oxygen, before the gas went global.

It was a critical moment in the evolution of life on Earth. Once organisms had acquired the ability to survive in the presence of oxygen, they could evolve to harness its chemical energy, and become the world's first oxygen-breathers.

http://bit.ly/1nGBJLI

Salmonella's Favorite Food Could Be Its Achilles' Heel

Salmonella's primary fuel source is the molecule fructose-asparagine. Starving it of that fuel in an infected person could kill it without harming beneficial gut bacteria.

Karen Hopkin reports

Download MP3

Summer's here and with it come picnics, barbecues and of course salmonella. The germ is notorious for contaminating a variety of favorite warm-weather foods. But the bacteria's palate is more limited than ours. Once salmonella makes its way into your system, it relies on a single unusual nutrient to survive. That's according to a study in the journal PLoS Pathogens. *[Mohamed M. Ali et al, Fructose-Asparagine Is a Primary Nutrient during Growth of Salmonella in the Inflamed Intestine]*

Most people tough it out when they get food poisoning from salmonella. That's because treatment with antibiotics would eliminate the infection, but also get rid of the gut bacteria that promote good health.

To figure out how to target salmonella specifically, researchers screened for genes vital for the microbe's survival during the active phase of infection. And they identified a cluster of five genes that work together to allow the bacteria to digest a molecule called fructose-asparagine. No other organisms are known to use this chemical for fuel, so starving salmonella of it could be a new strategy for fighting this foodborne bug while leaving desirable intestinal inhabitants unharmed.

Next, the researchers plan to see which foods contain large amounts of salmonella's go-to snack. But please, don't send unsolicited samples of Aunt Agnes's egg salad.

http://www.eurekalert.org/pub_releases/2014-07/sdsu-ndg071814.php

Newly discovered gut virus lives in half the world's population

Biologists at SDSU have found a previously unknown virus that is extremely widespread and could play a major role in obesity and diabetes

Odds are, there's a virus living inside your gut that has gone undetected by scientists for decades. A new study led by researchers at San Diego State University has found that more than half the world's population is host to a newly described virus, named crAssphage, which infects one of the most common types of gut bacteria, Bacteroidetes. This phylum of bacteria is thought to be connected with obesity, diabetes and other gut-related diseases. The research appears today in Nature Communications.

Robert A. Edwards, a bioinformatics professor at SDSU, and his colleagues stumbled upon the discovery quite by accident. Working with visiting researcher and corresponding author on the study Bas E. Dutilh, now at Radboud University Medical Center in The Netherlands, the researchers were using results from previous studies on gut-inhabiting viruses to screen for new viruses.

In the DNA fecal samples from 12 different individuals, they noticed a particular cluster of viral DNA, about 97,000 base pairs long, that the samples all had in common. When Edwards and his colleagues checked this discovery against a comprehensive listing of known viruses, they came up empty.

The researchers then screened for the virus across the database of the National Institute of Health's Human Microbiome Project (HMP), and Argonne National Laboratory's MG-RAST database, and again found it in abundance in samples derived from human feces.

To prove that the viral DNA they discovered in their computer data actually exists in nature, fellow SDSU virologist John Mokili used a technique known as DNA amplification to locate the virus in the original samples used to build NIH's database. "So we have a biological proof that the virus they found with the computer actually exists in the samples," Mokili said.

This was a new virus that about half the sampled people had in their bodies that nobody knew about. "It's not unusual to go looking for a novel virus and find one," Edwards said. "But it's very unusual to find one that so many people have in common. The fact that it's flown under the radar for so long is very strange."

An ancient virus

The fact that it's so widespread indicates that it probably isn't a particularly young virus, either. "We've basically found it in every population we've looked at,"

Edwards said. “As far as we can tell, it’s as old as humans are.” He and his team named the virus crAssphage, after the cross-assembly software program used to discover it.

Some of the proteins in crAssphage’s DNA are similar to those found in other well-described viruses. That allowed Edwards’ team to determine that their novel virus is one known as a bacteriophage, which infects and replicates inside bacteria - and using innovative bioinformatic techniques, they predicted that this particular bacteriophage proliferates by infecting a common phylum of gut bacteria known as Bacteriodetes.

Gut punch

Bacteriodetes bacteria live toward the end of the intestinal tract, and they are suspected to play a major role in the link between gut bacteria and obesity. What role crAssphage plays in this process will be a target of future research.

Further details about crAssphage have been difficult to come by. It’s unknown how the virus is transmitted, but the fact that it was not found in very young infants’ fecal samples suggests that it is not passed along maternally, but acquired during childhood. The makeup of the viral DNA suggests that it’s circular in structure. Further laboratory work has confirmed that the viral DNA is a singular entity, but it’s proven difficult to isolate.

“We know it’s there, but we can’t capture it quite yet,” Edwards said.

Once the virus is isolated, he hopes to delve into its role in obesity. It’s possible the virus in some way mediates the activity of Bacteriodetes colonies, but whether crAssphage promotes or suppresses obesity-related processes in the gut remains to be seen.

The virus might also be used to prevent or mitigate other diseases affected by the gut such as diabetes and gastroenterological maladies.

Once these processes are better understood, Edwards envisions one day the possibility of personalized medicine based on this virus.

“This could be a key to personalized phage medicine,” he said. “In individuals, we could isolate your particular strain of the virus, manipulate it to target harmful bacteria, then give it back to you.”

Key Collaborators

In addition to Edwards, SDSU researchers Katelyn McNair, Savannah Sanchez, Genivaldo G.Z. Silva, Lance Boling, Jeremy J. Barr, Victor Seguritan, Ben Felts, and Elizabeth A. Dinsdale worked on the project, in collaboration with Argonne National Laboratory in Illinois. The study’s corresponding author, Bas E. Dutilh, shares an affiliation with SDSU, Radboud University Medical Center in The Netherlands, and the Federal University of Rio de Janeiro in Brazil. Contributing researcher Ramy K. Aziz shares an affiliation with SDSU and Cairo University in Egypt. Contributing researcher Noriko Cassman was at SDSU during the time of the study and now is at the Netherlands Institute of Ecology.

http://phys.org/news/2014-07-billion-year-old-chemistry-cells-today.html

Four billion-year-old chemistry in cells today

Parts of the primordial soup in which life arose have been maintained in our cells today according to scientists at the University of East Anglia.

Research published today in the Journal of Biological Chemistry reveals how cells in plants, yeast and very likely also in animals still perform ancient reactions thought to have been responsible for the origin of life – some four billion years ago.

The primordial soup theory suggests that life began in a pond or ocean as a result of the combination of metals, gases from the atmosphere and some form of energy, such as a lightning strike, to make the building blocks of proteins which would then evolve into all species.

The new research shows how small pockets of a cell – known as mitochondria – continue to perform similar reactions in our bodies today.

These reactions involve iron, sulfur and electro-chemistry and are still important for functions such as respiration in animals and photosynthesis in plants.

Lead researcher Dr Janneke Balk, from UEA's school of Biological Sciences and the John Innes Centre, said: "Cells confine certain bits of dangerous chemistry to specific compartments of the cell.

"For example small pockets of a cell called mitochondria deal with electrochemistry and also with toxic sulfur metabolism. These are very ancient reactions thought to have been important for the origin of life.

"Our research has shown that a toxic sulfur compound is being exported by a mitochondrial transport protein to other parts of the cell. We need sulfur for making iron-sulfur catalysts, again a very ancient chemical process.

"The work shows that parts of the primordial soup in which life arose has been maintained in our cells today, and is in fact harnessed to maintain important biological reactions."

The research was carried out at UEA and JIC in collaboration with Dr Hendrik van Veen at the University of Cambridge. It was funded by the Biotechnology and Biological Sciences Research Council (BBSRC).

'A Conserved Mitochondrial ATB-Binding Cassette Transporter Exports Glutathione Polysufide for Cytosolic Metal Cofactor Assembly' is published in the Journal of Biological Chemistry.

More information: "A Conserved Mitochondrial ATP-Binding Cassette Transporter Exports Glutathione Polysulfide for Cytosolic Metal Cofactor Assembly." Schaedler TA, et al. J Biol Chem. 2014 Jul 8. pii: jbc.M114.553438. [Epub ahead of print]
www.ncbi.nlm.nih.gov/pubmed/25006243

http://www.eurekalert.org/pub_releases/2014-07/ltu-aii072414.php

Artificial intelligence identifies the musical progression of the Beatles

An artificial intelligence algorithm that can analyze and compare musical styles

Music fans and critics know that the music of the Beatles underwent a dramatic transformation in just a few years, but until now there hasn't been a scientific way to measure the progression. That could change now that computer scientists at Lawrence Technological University have developed an artificial intelligence algorithm that can analyze and compare musical styles, enabling research into the musical progression of the Beatles.

Assistant Professor Lior Shamir and graduate student Joe George had previously developed audio analysis technology to study the vocal communication of whales, and they expanded the algorithm to analyze the albums of the Beatles and other well-known bands such as Queen, U2, ABBA and Tears for Fears. The study, published in the August issue of the journal Pattern Recognition Letters, demonstrates scientifically that the structure of the Beatles music changes progressively from one album to the next.

The algorithm works by first converting each song to a spectrogram – a visual representation of the audio content. That turns an audio analysis task into an image analysis problem, which is solved by applying comprehensive algorithms that turn each music spectrogram into a set of almost 3,000 numeric descriptors reflecting visual aspects such as textures, shapes and the statistical distribution of the pixels. Pattern recognition and statistical methods are then used to detect and quantify the similarities between different pieces of music.

In popular music, albums are widely considered milestones in the stylistic development of music artists, and these collections of songs provide a convenient unit for establishing measurements to quantify a band's progression.

LTU's study analyzed 11 songs from each of the 13 Beatles studio albums released in Great Britain, and quantified the similarities between each song and all the others in the study. The results for the individual songs were then used to compare the similarities between the albums.

The automatic placement of the albums by the algorithm was in agreement with the chronological order of the recording of each album, starting with the Beatles' first album, "Please, Please Me," and followed by the subsequent early albums, "With the Beatles," "Beatles for Sale" and "A Hard Day's Night."

The automatic association of these albums demonstrated that the computer algorithm determined that the songs on the first album, "Please, Please Me," were most like the group of songs on the second album, "With the Beatles," and least like the songs on the last album recorded, "Abbey Road."

The algorithm then placed the albums "Help!," and "Rubber Soul," followed by "Revolver," "Sergeant Pepper's Lonely Hearts Club Band," "Magical Mystery Tour," "Yellow Submarine," and "The Beatles" (The White Album).

"Let It Be" was the last album released by the Beatles, but the algorithm correctly identified those songs as having been recorded earlier than the songs on "Abbey Road."

"People who are not Beatles fans normally can't tell that 'Help!' was recorded before 'Rubber Soul,' but the algorithm can," Shamir said. "This experiment demonstrates that artificial intelligence can identify the changes and progression in musical styles by 'listening' to popular music albums in a completely new way."

The computer algorithm was able to deduce the chronological order of the albums of the other groups in the study by analyzing the audio data alone – with one notable exception. Strong similarities were identified between two Tears for Fears albums released 15 years apart. That makes sense because "Seeds of Love," released in 1989, was the last album before the band's breakup, and "Everybody Loves a Happy Ending," released in 2004, was recorded after the band reunited. Those two albums had less in common with two solo albums released by Roland Orzabal, the group's principal songwriter, after the band split up in 1991.

In the case of "Queen," the computer not only sorted the albums by their chronological order, but also distinguished between albums before and after the album "Hot Space," which represented a major shift in Queen's musical style.

In this era of big data, such algorithms can assist in searching, browsing, and organizing large music databases, as well as identifying music that matches an individual listener's musical preferences.

In the case of the Beatles, Shamir believes this type of research will have historical significance. "The baby boomers loved the music of the Beatles, I love the Beatles, and now my daughters and their friends love the Beatles. Their music will live on for a very long time," Shamir said. "It is worthwhile to study what makes their music so distinctive, and computer science and big data can help."

http://www.eurekalert.org/pub_releases/2014-07/uoo-8oo072314.php

8.2 percent of our DNA is 'functional'

Only 8.2% of human DNA is likely to be doing something important – is 'functional' – say Oxford University researchers.

This figure is very different from one given in 2012, when some scientists involved in the ENCODE (Encyclopedia of DNA Elements) project stated that 80% of our genome has some biochemical function. That claim has been controversial, with many in the field arguing that the biochemical definition of

'function' was too broad – that just because an activity on DNA occurs, it does not necessarily have a consequence; for functionality you need to demonstrate that an activity matters.

To reach their figure, the Oxford University group took advantage of the ability of evolution to discern which activities matter and which do not. They identified how much of our genome has avoided accumulating changes over 100 million years of mammalian evolution – a clear indication that this DNA matters, it has some important function that needs to be retained.

'This is in large part a matter of different definitions of what is "functional" DNA,' says joint senior author Professor Chris Pointing of the MRC Functional Genomics Unit at Oxford University. 'We don't think our figure is actually too different from what you would get looking at ENCODE's bank of data using the same definition for functional DNA.

'But this isn't just an academic argument about the nebulous word "function". These definitions matter. When sequencing the genomes of patients, if our DNA was largely functional, we'd need to pay attention to every mutation. In contrast, with only 8% being functional, we have to work out the 8% of the mutations detected that might be important. From a medical point of view, this is essential to interpreting the role of human genetic variation in disease.'

The researchers Chris Rands, Stephen Meader, Chris Ponting and Gerton Lunter report their findings in the journal PLOS Genetics. They were funded by the UK Medical Research Council and the Wellcome Trust.

The researchers used a computational approach to compare the complete DNA sequences of various mammals, from mice, guinea pigs and rabbits to dogs, horses and humans.

Dr Gerton Lunter from the Wellcome Trust Centre for Human Genetics at Oxford University, the other joint senior author, explained: 'Throughout the evolution of these species from their common ancestors, mutations arise in the DNA and natural selection counteracts these changes to keep useful DNA sequences intact.'

The scientists' idea was to look at where insertions and deletions of chunks of DNA appeared in the mammals' genomes. These could be expected to fall approximately randomly in the sequence – except where natural selection was acting to preserve functional DNA, where insertions and deletions would then lie further apart.

'We found that 8.2% of our human genome is functional,' says Dr Lunter. 'We cannot tell where every bit of the 8.2% of functional DNA is in our genomes, but our approach is largely free from assumptions or hypotheses. For example, it is not dependent on what we know about the genome or what particular experiments are used to identify biological function.'

The rest of our genome is leftover evolutionary material, parts of the genome that have undergone losses or gains in the DNA code – often called 'junk' DNA.

'We tend to have the expectation that all of our DNA must be doing something. In reality, only a small part of it is,' says Dr Chris Rands, first author of the study and a former DPhil student in the MRC Functional Genomics Unit at Oxford University.

Not all of the 8.2% is equally important, the researchers explain. A little over 1% of human DNA accounts for the proteins that carry out almost all of the critical biological processes in the body. The other 7% is thought to be involved in the switching on and off of genes that encode proteins – at different times, in response to various factors, and in different parts of the body. These are the control and regulation elements, and there are various different types.

'The proteins produced are virtually the same in every cell in our body from when we are born to when we die,' says Dr Rands. 'Which of them are switched on, where in the body and at what point in time, needs to be controlled – and it is the 7% that is doing this job.'

In comparing the genomes of different species, the researchers found that while the protein-coding genes are very well conserved across all mammals, there is a higher turnover of DNA sequence in the regulatory regions as this sequence is lost and gained over time.

Mammals that are more closely related have a greater proportion of their functional DNA in common. But only 2.2% of human DNA is functional and shared with mice, for example – because of the high turnover in the regulatory DNA regions over the 80 million years of evolutionary separation between the two species.

'Regulatory DNA evolves much more dynamically that we thought,' says Dr Lunter, 'but even so, most of the changes in the genome involve junk DNA and are irrelevant.' He explains that although there is a lot of functional DNA that isn't shared between mice and humans, we can't yet tell what is novel and explains our differences as species, and which is just a different gene-switching system that achieves the same result.

Professor Ponting agrees: 'There appears to be a lot of redundancy in how our biological processes are controlled and kept in check. It's like having lots of different switches in a room to turn the lights on. Perhaps you could do without some switches on one wall or another, but it's still the same electrical circuit.'

He adds: 'The fact that we only have 2.2% of DNA in common with mice does not show that we are so different. We are not so special. Our fundamental biology is very similar. Every mammal has approximately the same amount of functional DNA, and approximately the same distribution of functional DNA that is highly

important and less important. Biologically, humans are pretty ordinary in the scheme of things, I'm afraid. 'I'm definitely not of the opinion that mice are bad model organisms for animal research. This study really doesn't address that issue,' he notes.

This article is available at:
http://www.plosgenetics.org/article/info:doi/10.1371/journal.pgen.1004525
The study was funded by UK Medical Research Council and the Wellcome Trust.

http://www.eurekalert.org/pub_releases/2014-07/wsu-plt072114.php

Pesticide linked to 3 generations of disease

Methoxychlor causes epigenetic changes

PULLMAN, Wash. – Washington State University researchers say ancestral exposures to the pesticide methoxychlor may lead to adult onset kidney disease, ovarian disease and obesity in future generations.

"What your great-grandmother was exposed to during pregnancy, like the pesticide methoxychlor, may promote a dramatic increase in your susceptibility to develop disease, and you will pass this on to your grandchildren in the absence of any continued exposures," says Michael Skinner, WSU professor and founder of its Center for Reproductive Biology.

He and his colleagues document their findings in a paper published online in PLOS ONE. The study was funded by the National Institutes of Health.

DDT replacement banned in 2003

Methoxychlor - also known as Chemform, Methoxo, Metox or Moxie - was introduced in 1948 and widely used during the 1970s as a safer replacement for DDT. It was used on crops, ornamental plants, livestock and pets. It is still used in many countries around the world.

It was banned in the U.S. in 2003 due to its toxicity and ability to disrupt endocrine systems. Methoxychlor can behave like the hormone estrogen and profoundly affects the reproductive system.

Supports earlier epigenetic findings

When Skinner and his colleagues exposed gestating rats to methoxychlor at a range typical of high environmental exposures, they saw increases in the incidence of kidney disease, ovary disease and obesity in offspring spanning three generations. The incidence of multiple diseases increased in the third generation or "great-grandchildren."

The researchers say the pesticide may be affecting how genes are turned on and off in the progeny of an exposed animal, even though its DNA and gene sequences remain unchanged.

This is called transgenerational epigenetic inheritance. In recent years, the Skinner lab has documented epigenetic effects from a host of environmental toxicants, including DDT, plastics, pesticides, fungicides, dioxins, hydrocarbons and the plasticizer bisphenol-A or BPA. The newest findings support those observations.

Implications for obesity, fertility, disease

The work is also the first to show that a majority of transgenerational disease traits can be transmitted primarily through the female line.

Additionally, the study identified mutations in the sperm epigenome of great-grandchild male rats. The epigenome functions like a set of switches for regulating gene expression and can be altered by environmental conditions.

The epigenetic changes observed were specific to methoxychlor exposure and may prove to be valuable biomarkers for future research on transgenerational disease.

For people exposed to the pesticide, Skinner says his findings have implications such as reduced fertility, increased adult onset disease and the potential to pass on those conditions to subsequent generations.

He suggests that ancestral exposures to methoxychlor over the past 50 years in North America may play a part in today's increasing rates of obesity and disease.

http://www.eurekalert.org/pub_releases/2014-07/asfm-tmm072414.php

The microbes make the sake brewery

First time investigators have taken a microbial census of a sake brewery

A sake brewery has its own microbial terroir, meaning the microbial populations found on surfaces in the facility resemble those found in the product, creating the final flavor according to research published ahead of print in the journal Applied and Environmental Microbiology. This is the first time investigators have taken a microbial census of a sake brewery.

Many sake makers inoculate with both bacteria and yeast, says corresponding author David A. Mills of the University of California, Davis, but he and his colleagues investigated a sake brewery where inoculation is restricted to a single species, Aspergillus oryzae, at the first of three stages of fermentation.

"The purpose was to be able to ask the question, 'do the environmental surfaces have microbiota that are similar to those that normally are added to ferment the product?'" says Mills.

And despite the single stage one inoculation, the microbial populations change dramatically at each fermentation stage - koji, moto, and moromi.

"The kojii fermentation is dominated by an inoculated fungus, Aspergillus oryzae, which helps process the rice into smaller, more available sugars," says Mills. "The Kojii is then diluted with steamed rice and water to form the seed mash or moto. In this stage the alcoholic fermentation commences with yeast and various lactic acid-producing bacteria populations expanding."

That, says Mills, is followed by the major fermentation in sake. "Yeast perform the alcoholic fermentation, while a range of other bacteria - Bacillus, Staphylococcus, Lactobacillus - consume available nutrients and stabilize the product."

"At each stage, most of these organisms - with the exception of the added A. oryzae - could also be found on the equipment surfaces, suggesting the house microbiome provides the necessary microbes to carry out the fermentation," says Mills. "Thus, the environmental conditions are important for controlling these fermentations."

The results echo those of studies Mills and collaborators have done on other food facilities: an artisanal cheese maker, and wine facilities, he says. He adds that this line of research is currently at the natural history stage where census is taken, and that ecological understanding, the kind of understanding that will enable predictive product improvement, will come later. But he expects this kind of facility monitoring to become the norm.

"Understanding the microbial interface between food facilities and food products in a global way will be important for controlling the safety and quality of many different foods and beverages," says Mills.

The manuscript can be found online at http://bit.ly/asmtip0714h. The final version of the article is scheduled for the September 2014 issue of Applied and Environmental Microbiology.

http://phys.org/news/2014-07-fukushima-monkeys-effects.html

Fukushima monkeys show possible 'effects of radiation'

Monkeys near the stricken Fukushima nuclear power plant have lower blood cell counts than cousins living further away, possibly because of radiation exposure, a study said Thursday.

A Japanese research team wrote in the journal Nature Scientific Reports that although they could not prove the link, the blood levels "might likely be the result of exposure to some form of radioactive material".

Fewer blood cells could make the monkeys more prone to disease, they said, and "may suggest that the immune system has been compromised to some extent".

The team had compared white and red blood cell levels in macaques living in a forest area of Fukushima City, 70 kilometres (43 miles) from the nuclear plant, with that of 31 monkeys living 400 km away in the Shimokita Peninsula.

"Compared with Shimokita monkeys, Fukushima monkeys had significantly low white and red blood cell counts," said the researchers.

The study sought to examine the health effects of long-term radioactive exposure on wild Japanese macaques following the massive earthquake and nuclear meltdown at Fukushima in March 2011.

Such data from non-human primates, our closest relatives, could contribute to knowledge about the health effects of radiation exposure on humans, the team said.

But some commentators criticised the research method. Jim Smith, environmental science professor at the University of Portsmouth in Britain, said the dosage inferred in the study was unlikely to have had a significant effect on the monkeys' blood cell count. "I think it much more likely that the apparently low blood cell counts in the Fukushima monkeys are caused by something other than radiation."

Geraldine Thomas, a professor of molecular pathology at Imperial College London, added the radiation doses would have been less than a person would receive on a flight from London to Tokyo. The blood cell count may be caused by other factors such as a new diet or other environmental changes brought on by the tsunami, she said.

http://phys.org/news/2014-07-nike-krypton-laser-guinness-world.html

Nike krypton laser achieves spot in Guinness World Records

"Highest Projectile Velocity" of greater than 1,000 kilometers per second (km/s), a speed equivalent to two-and-a-quarter million miles per hour.

A set of experiments conducted on the Nike krypton fluoride (KrF) laser at the U.S. Naval Research Laboratory (NRL) nearly five years ago has, at long last, earned the coveted Guinness World Records title for achieving "Highest Projectile Velocity" of greater than 1,000 kilometers per second (km/s), a speed equivalent to two-and-a-quarter million miles per hour.

The previous record was held by researchers at Osaka University's Institute of Laser Engineering in Japan, who in 2006 used a neodymium glass (Nd:glass) laser to accelerate a target to 700 km/s. The record, currently held by NRL, was achieved in collaboration with the NRL Plasma Physics Division and the group from Japan, demonstrating the advantages of the high uniformity and short wavelength of the KrF laser technology.

"The impact of the highly accelerated target on a stationary foil generated thermonuclear fusion neutrons whose energy spread indicated that a gigabar - that's the pressure of a billion atmospheres - was achieved in the collision," said Dr. Max Karasik, NRL Laser Plasma Branch. "The results highlight the advantages of a krypton-fluoride laser in efficiently generating uniform pressures required for fuel compression in inertial confinement fusion."

In the experiments, thin plastic foils were accelerated to 1,000 km/s over a distance of less than a millimeter. The moving foils then collided with a stationary foil, generating thermo-nuclear temperatures and neutrons from fusion reactions. The high ablative pressure applied to compress and accelerate targets is used in inertial confinement fusion and high energy density research.

NRL received the official Guinness World Records certificate, February 2014, with distinction given to the research that "…probe[s] possibilities for future clean-energy sources." However, since the 2009 experiment, Karasik says NRL has raised the bar. With an improved laser pulse shape, researchers at the Nike laser facility have reached target velocities of 1,180 km/s.

http://www.bbc.com/news/science-environment-28407381

Fluffy and feathery' dinosaurs were widespread

All dinosaurs were covered with feathers or had the potential to grow feathers, a study suggests.

By Pallab Ghosh Science correspondent, BBC News

The discovery of 150-million-year-old fossils in Siberia indicates that feathers were much more widespread among dinosaurs than previously thought.

The find "has completely changed our vision of dinosaurs", the lead researcher told BBC News. The details have been published in the journal Science.

The creature, called Kulindadromeus zabaikalicus, was about 1m long, with a short snout, long hind legs, short arms, and five strong fingers. Its teeth show clear adaptations for chewing plants. Until now, fossilised evidence of feathery dinosaurs has come from China and from a meat eating group called theropods.

The latest discovery, in Russia, is from a completely separate group of plant-eating dinosaurs called ornithischians - which account for half of all dinosaurs.

Fluffy covering

The find takes the origin of feathers millions of years further back in time than had previously been thought, said Dr Pascal Godefroit of the Royal Belgian Institute of Natural Sciences in Brussels, Belgium, who led the research.

"It was a big surprise," he said. "The fact that feathers have now been discovered in two distinct groups, theropods in China and ornithischians in Russia means that the common ancestor of these species which might have existed 220 million years ago also probably had feathers."

The discovery has "completely changed our vision of dinosaurs", he added.

"Instead of thinking of dinosaurs as dry, scary scaly creatures a lot of them actually had a fluffy, downy covering like feathers on a chick," said co-researcher Dr Maria McNamara of Cork University in Ireland.

Alternative view

So do all the pictures of dinosaurs in children's books need to be redrawn to make creatures like Triceratops, Stegosaurus, Tyrannosaurus rex and the vicious Velociraptor, fluffier and cuter?

Perhaps a little bit, according to Professor Mike Benton, of Bristol University, who was also involved in the work. "Our research doesn't mean that all dinosaurs had feathers, especially as adults," he told BBC News.

"Some will have had feathers as young animals and kept them throughout their lives. Others may have lost feathers as they grew up, and became large enough not to need them, or replaced feathers with scales or relied on bony plates in the skin for protection."

The key point is that dinosaurs were all initially feathered and warm blooded, confirmation of an idea that has prevailed for years, he said. "Feathers were used first for insulation and signalling; they only later became adapted for flight."

But Dr Paul Barrett of the Natural History Museum in London, has doubts. "Most feathers have a branching structure," he told BBC News. "Instead these look like little streamers coming from a central plate. No bird has that structure in any part of its plumage and none of the developmental models that biologists use to understand the evolution of feathers includes a stage that has anything like that kind of anatomy."

http://www.eurekalert.org/pub_releases/2014-07/tu-tda072214.php

Total darkness at night is key to success of breast cancer therapy -- Tulane study

Light at night shuts off nocturnal production of melatonin, rendering breast cancer completely resistant to tamoxifen

Exposure to light at night, which shuts off nighttime production of the hormone melatonin, renders breast cancer completely resistant to tamoxifen, a widely used breast cancer drug, says a new study by Tulane University School of Medicine cancer researchers. The study, "Circadian and Melatonin Disruption by Exposure to Light at Night Drives Intrinsic Resistance to Tamoxifen Therapy in Breast Cancer," published in the journal Cancer Research, is the first to show that melatonin is vital to the success of tamoxifen in treating breast cancer.

Principal investigators and co-leaders of Tulane's Circadian Cancer Biology Group, Steven Hill and David Blask, along with team members Robert Dauchy and Shulin Xiang, investigated the role of melatonin on the effectiveness of tamoxifen in combating human breast cancer cells implanted in rats.

"In the first phase of the study, we kept animals in a daily light/dark cycle of 12 hours of light followed by 12 hours of total darkness (melatonin is elevated during the dark phase) for several weeks," says Hill. "In the second study, we exposed them to the same daily light/dark cycle; however, during the 12 hour dark phase, animals were exposed to extremely dim light at night (melatonin levels are suppressed), roughly equivalent to faint light coming under a door."

Melatonin by itself delayed the formation of tumors and significantly slowed their growth but tamoxifen caused a dramatic regression of tumors in animals with

either high nighttime levels of melatonin during complete darkness or those receiving melatonin supplementation during dim light at night exposure.

These findings have potentially enormous implications for women being treated with tamoxifen and also regularly exposed to light at night due to sleep problems, working night shifts or exposed to light from computer and TV screens.

"High melatonin levels at night put breast cancer cells to 'sleep' by turning off key growth mechanisms. These cells are vulnerable to tamoxifen. But when the lights are on and melatonin is suppressed, breast cancer cells 'wake up' and ignore tamoxifen," Blask says.

The study could make light at night a new and serious risk factor for developing resistance to tamoxifen and other anticancer drugs and make the use of melatonin in combination with tamoxifen, administered at the optimal time of day or night, standard treatment for breast cancer patients.

http://bit.ly/1k0UgTT

Earth survived near-miss from 2012 solar storm: NASA

Back in 2012, the Sun erupted with a powerful solar storm that just missed the Earth but was big enough to "knock modern civilization back to the 18th century," NASA said.

The extreme space weather that tore through Earth's orbit on July 23, 2012, was the most powerful in 150 years, according to a statement posted on the US space agency website Wednesday. However, few Earthlings had any idea what was going on. "If the eruption had occurred only one week earlier, Earth would have been in the line of fire," said Daniel Baker, professor of atmospheric and space physics at the University of Colorado.

Instead the storm cloud hit the STEREO-A spacecraft, a solar observatory that is "almost ideally equipped to measure the parameters of such an event," NASA said. Scientists have analyzed the treasure trove of data it collected and concluded that it would have been comparable to the largest known space storm in 1859, known as the Carrington event. It also would have been twice as bad as the 1989 solar storm that knocked out power across Quebec, scientists said.

"I have come away from our recent studies more convinced than ever that Earth and its inhabitants were incredibly fortunate that the 2012 eruption happened when it did," said Baker.

The National Academy of Sciences has said the economic impact of a storm like the one in 1859 could cost the modern economy more than two trillion dollars and cause damage that might take years to repair.

Experts say solar storms can cause widespread power blackouts, disabling everything from radio to GPS communications to water supplies - most of which rely on electric pumps.

They begin with an explosion on the Sun's surface, known as a solar flare, sending X-rays and extreme UV radiation toward Earth at light speed. Hours later, energetic particles follow and these electrons and protons can electrify satellites and damage their electronics. Next are the coronal mass ejections, billion-ton clouds of magnetized plasma that take a day or more to cross the Sun-Earth divide. These are often deflected by Earth's magnetic shield, but a direct hit could be devastating.

There is a 12 percent chance of a super solar storm the size of the Carrington event hitting Earth in the next 10 years, according to physicist Pete Riley, who published a paper in the journal Space Weather earlier this year on the topic. His research was based on an analysis of solar storm records going back 50 years.

"Initially, I was quite surprised that the odds were so high, but the statistics appear to be correct," said Riley. "It is a sobering figure."

http://phys.org/news/2014-07-saltwater-fracking-ocean.html

'Saltwater' from fracking spill much different from ocean water

Fracking fluids bear little resemblance to what's found in the ocean

In early July, a million gallons of salty drilling waste spilled from a pipeline onto a steep hillside in western North Dakota's Fort Berthold Reservation. The waste - a byproduct of oil and gas production - has now reached a tributary of Lake Sakakawea, which provides drinking water to the reservation.

The oil industry called the accident a "saltwater" spill. But the liquid that entered the lake bears little resemblance to what's found in the ocean.

The industry's wastewater is five to eight times saltier than seawater, said Bill Kappel, a hydrogeologist emeritus at the U.S. Geological Survey. It's salty enough to sting the human tongue, and contains heavy metals in concentrations that might not meet drinking water standards. The briny mix can also include radioactive material. Heavy metals and radioactive materials are toxic at certain concentrations.

"You don't want to be drinking this stuff," Kappel said.

The North Dakota spill has killed vegetation and contaminated the soil, and cleanup crews are working on remediation and monitoring. In an email, a representative of Crestwood Midstream Partners - the parent company of Arrow Pipelines, the company responsible for the spill - said there is "no evidence of an impact to the local water supply."

Confusion persists over the wastewater's environmental and health effects because little is known about the composition of the spilled waste. The compounds it contains vary widely depending on local geology and drilling practices. And there are inconsistencies even within the industry over the definition of "saltwater," which may or may not contain hydraulic fracturing (fracking) fluids.

The "terms are used very loosely, probably on purpose," Kappel said.
Jim Ladlee, associate director of the Penn State Marcellus Center for Outreach and Research, said oilfield definitions vary by company, and the same operator may use different words for the same waste product in different parts of the country.
Both Ladlee and Kappel said it's impossible to understand the potential impact in North Dakota without additional information about what, exactly, was in the pipeline.
The Crestwood representative did not answer questions about the saltwater composition. Because the spill occurred on tribal land, Alison Ritter, a spokeswoman at the North Dakota Department of Mineral Resources, directed all questions to the Mandan, Hidatsa and Arikara Nation, which did not respond by deadline.
Here's what is known:
The industry-coined term saltwater usually refers to three types of waste.
1.) The naturally occurring brines located in oil and gas formations hundreds or thousands of feet underground, known as produced water. The brines consist of water and dissolved chemicals leached from the surrounding rock. These include:
-Sodium and chloride (the compounds that make up table salt).
-Heavy metals such as chromium, cobalt, nickel, copper, zinc, arsenic, selenium, silver, cadmium, antimony, mercury, thallium and lead.
-Radioactive material from buried rock.
-Other dissolved compounds such as barium, calcium and bromide.
Once a well begins operating, produced water flows out of the wellbore along with the oil and/or gas. The mixture is separated at the surface, and the produced water is trucked or piped away for disposal through "saltwater pipelines."
2.) A mixture of produced water and fracking fluids, called flowback. Fracking fluids contain millions of gallons of water, millions of pounds of sand and thousands of gallons of chemical additives, some of which are toxic.
Flowback comes out of the well during the first two to three weeks after it's fracked. Like produced water, the flowback is shipped via pipeline for disposal in injection wells or waste pits.
3.) Produced water, which has been treated to remove almost everything but salt and is reused in the fracking process. Ladlee, the Marcellus Outreach representative, said produced water is sent to treatment plants, which remove most of the dissolved material except for the salt. The treated salty mixture is diluted with fresh water, and fracking chemicals are added to create a new batch of fracking fluid, which is used to frack another well. If the North Dakota pipeline was carrying fluid from a treatment facility to a well site for recycling, Ladlee said, then the saltwater would contain very salty water, but with few heavy metals and radioactive material.

The North Dakota spill caught media attention because it was unusually large. But smaller "saltwater" spills occur more frequently. According to The Associated Press, there were 74 such spills in North Dakota last year, spilling a total of 924,000 gallons.

http://bit.ly/1l32JkA

Prototype display uses eyeglass prescription to allow for viewing devices without glasses

An experimental display technology being developed by Microsoft, U.C. Berkeley and MIT aims to allow users with vision problems to clearly see device screens without the need for glasses.

The technology is based on an algorithm developed by the team that accepts a person's eyeglass prescription and uses it to alter the image projected by a smartphone, tablet, computer, etc. allowing for viewing without eyeglasses.
The display technology has two parts, the first involves using an algorithm run on the device to convert eyeglass information to a change in the way light is generated by individual pixels on a screen. The second part is an acrylic light filter laid over the display - it has tiny holes in it, each of which sit directly over a pixel. Together the altered pixels and filter produce an image on the display that mimics what a user would see on a normal screen if they were wearing the same prescription glasses.
The team has built a prototype of just such a system using an iPod Touch smartphone and cameras that are able to simulate vision problems. They note that while the system they've developed thus far works in principle, there is still a lot of work to do before it could be implemented as a commercial product. Currently, the prototype only works when viewed from a set distance, movement by the person viewing the display would result in distortion. The researchers envision an addition to the system that monitors the location of the head and eyes of the person doing the viewing, and adjusts the display in real-time. Another problem is that the display only allows one person (the one whose prescription has been used) to view the device's screen clearly. Thus, it wouldn't really work for a television screen, at least as its configured now. The team believes they could make their technology work for multiple users when applied to higher pixel density devices.
In addition to allowing people to view their devices without their glasses, the researchers note that it might open up new possibilities for people with other vision problems - those that have trefoil and spherical aberrations, for example.
The team will be presenting their prototype at this years' SIGGRAPH computer graphics conference next month in Vancouver.

http://www.eurekalert.org/pub_releases/2014-07/vumc-vse072514.php

Vanderbilt study examines bacteria's ability to fight obesity

A probiotic that prevents obesity could be on the horizon.

Bacteria that produce a therapeutic compound in the gut inhibit weight gain, insulin resistance and other adverse effects of a high-fat diet in mice, Vanderbilt University investigators have discovered.

"Of course it's hard to speculate from mouse to human," said senior investigator Sean Davies, Ph.D., assistant professor of Pharmacology. "But essentially, we've prevented most of the negative consequences of obesity in mice, even though they're eating a high-fat diet."

Regulatory issues must be addressed before moving to human studies, Davies said, but the findings published in the August issue of the Journal of Clinical Investigation suggest that it may be possible to manipulate the bacterial residents of the gut - the gut microbiota - to treat obesity and other chronic diseases.

Davies has a long-standing interest in using probiotic bacteria - "friendly" bacteria like those in yogurt - to deliver drugs to the gut in a sustained manner, in order to eliminate the daily drug regimens associated with chronic diseases.

In 2007, he received a National Institutes of Health Director's New Innovator Award to develop and test the idea. "The NIH basically said, 'we like this idea, now make it work,'" Davies said. "The New Innovator Award was critical to our success."

Other studies have demonstrated that the natural gut microbiota plays a role in obesity, diabetes and cardiovascular disease. "The types of bacteria you have in your gut influence your risk for chronic diseases," Davies said. "We wondered if we could manipulate the gut microbiota in a way that would promote health."

To start, the team needed a safe bacterial strain that colonizes the human gut. They selected E. coli Nissle 1917, which has been used as a probiotic treatment for diarrhea since its discovery nearly 100 years ago.

They genetically modified the E. coli Nissle strain to produce a lipid compound called NAPE, which is normally synthesized in the small intestine in response to feeding. NAPE is rapidly converted to NAE, a compound that reduces both food intake and weight gain. Some evidence suggests that NAPE production may be reduced in individuals eating a high-fat diet. "NAPE seemed like a great compound to try - since it's something that the host normally produces," Davies said.

The investigators added the NAPE-producing bacteria to the drinking water of mice eating a high-fat diet for eight weeks. Mice that received the modified bacteria had dramatically lower food intake, body fat, insulin resistance and fatty liver compared to mice receiving control bacteria.

They found that these protective effects persisted for at least four weeks after the NAPE-producing bacteria were removed from the drinking water. And even 12 weeks after the modified bacteria were removed, the treated mice still had much lower body weight and body fat compared to the control mice. Active bacteria no longer persisted after about six weeks.

"We still haven't achieved our ultimate goal, which would be to do one treatment and then never have to administer the bacteria again," Davies said. "Six weeks is pretty long to have active bacteria, and the animals are still less obese 12 weeks out. "This paper provides a proof of concept," he said. "Clearly, we can get enough bacteria to persist in the gut and have a sustained effect. We would like for that effect to last longer."

Davies noted that the researchers also observed effects of the compounds in the liver, suggesting that it may be possible to use modified bacteria to deliver therapeutics beyond the gut.

The investigators are currently working on strategies to address regulatory issues related to containing the bacteria, for example by knocking out genes required for the bacteria to live outside the treated host.

Zhongyi Chen, M.D., Ph.D., and Lilu Guo, Ph.D., are co-first authors of the JCI paper. This research was supported by the New Innovator Award (OD003137) and by other grants from the National Institutes of Health (AT007830, DK059637, DK020593, RR024975, DK092993).

http://www.eurekalert.org/pub_releases/2014-07/acoe-nes072514.php

New EMS system in Arizona dramatically improves survival from cardiac arrest

A new system that sent patients to designated cardiac receiving centers dramatically increased the survival rate of victims of sudden cardiac arrest in Arizona, according to a study published online yesterday in Annals of Emergency Medicine.

WASHINGTON -- "We knew lives would be saved if the hospitals implemented the latest cutting edge guidelines for post-cardiac arrest care and we were able to get cardiac arrest patients to those hospitals, similar to what is done for Level 1 trauma patients," said lead study author Daniel Spaite, MD, Director of EMS Research at the University of Arizona Emergency Medicine Research Center in Phoenix and Tucson and a professor and distinguished chair of emergency medicine at the University of Arizona College of Medicine. "Taking these patients directly to a hospital optimally prepared to treat cardiac arrest gave patients a better chance of survival and of preventing neurologic damage, a common result of these cardiac events."

Under the study, 31 hospitals, serving about 80 percent of the state's population, were designated as cardiac receiving centers between December 2007 and

November 2010. Approximately 55 emergency medicine service agencies also participated in the study.

The study shows that the survival rate increased by more than 60 percent during the four-year period of the study, from 2007 to 2010. More importantly, when the results were adjusted for the various factors that significantly impact survival (such as age and how quickly the EMS system got to the patients after their arrest), the likelihood of surviving an arrest more than doubled. In addition, the likelihood of surviving with good neurological status also more than doubled.

This statewide effort was accomplished through the Save Hearts Arizona Registry and Education-SHARE Program, a partnership involving the Arizona Department of Health Services, the University of Arizona, over 30 hospitals and more than 100 fire departments and EMS agencies. The SHARE Program is part of a network of statewide cardiac resuscitation programs dedicated to improving cardiac arrest survival and working together as the HeartRescue Project.

"We worked closely with the hospitals around the state to implement these Guidelines and then formally recognized the hospitals as Cardiac Receiving Centers (CRCs) ," said Ben Bobrow, MD, Medical Director of the Bureau of Emergency Medicine Services and Trauma System for the Arizona Department of Health Services in Phoenix, Ariz. "We then developed protocols for our EMS agencies to transport post-cardiac arrest patients to those centers. Our overarching goal was to have more cardiac arrest victims leave the hospital in good shape and be able to return to their families and careers. As we suspected, 'regionalizing' the care for these critically-ill patients markedly increased their likelihood of survival and good neurologic outcome."

Dr. Bobrow, who is also a professor of emergency medicine at the University of Arizona College of Medicine-Phoenix and an emergency physician at Maricopa Medical Center, said the study shows that just transporting these patients to the nearest emergency department does not maximize the likelihood of a positive outcome. According to Dr. Bobrow, "Our data show that if the state approaches this as a regionalized system of emergency care delivery, significantly more people survive cardiac arrest and return to their families."

Dr. Spaite commented that dozens of cardiac arrest victims survived as a direct result of this effort in Arizona, and implementing similar regionalized post-cardiac arrest systems of care around the country "could potentially save thousands of lives each year from one of the leading causes of death."

The state began recognizing Cardiac Receiving Centers in 2007, and the following year began allowing EMS agencies to transport arrest victims to those centers as long as the increase in transport time to reach the receiving center was less than 15 minutes.

"This is the first statewide effort of its kind," said Dr. Bobrow. "While there have been individual or small groups of hospitals that have reported on implementing post-arrest care, widespread specialized regionalization, especially on a scope as large as a state, has not been previously reported anywhere in world. What happened in our state was very encouraging and exciting!"

http://phys.org/news/2014-07-widespread-seismic-sea-floor.html

US plans widespread seismic testing of sea floor

The U.S. government is planning to use sound blasting to conduct research on the ocean floor along most of the East Coast, using technology similar to that which led to a court battle by environmentalists in New Jersey.

AP - The U.S. Geological Survey plans to map the outer limits of the continental shelf and study underwater landslides that would help predict where and when tsunamis might occur. But environmentalists say it could cause the same type of marine life damage they fought unsuccessfully to prevent this month off New Jersey.

"New Jersey's marine life, fisheries and coastal economy can't get a break," said Cindy Zipf, executive director of Clean Ocean Action, which led the battle to block a sound blasting research plan.

Although it involves the same basic technology, the new plan is much wider-ranging. It would begin near the U.S.-Canadian offshore border and extend as far south as Florida.

John Haines, coordinator of the Geological Survey's coastal and marine geology program, said his research will be low-impact. It is designed to more precisely map the far reaches of the continental shelf to better determine where the United States' exclusive rights to undersea resources such as fish and shellfish extend.

It is not being done to map potential oil, gas or mineral deposits, he said. "As hard as it is to believe, we don't know in the U.S. where on the seabed our right to protect and use resources ends," he said. Data from the study also could show which areas of the U.S. and Caribbean coasts could be vulnerable to tsunamis.

The Geological Survey study is due to run for about three weeks sometime between August and September this year, and a similar period next year, Haines said. Zipf said researchers would blast the ocean floor with sound waves measuring from 236 to 265 decibels every 20 to 24 seconds for at least 17 days each year of the survey.

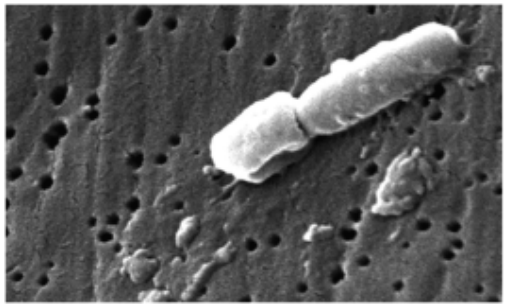
Environmentalists say the noise could harm or even kill marine life including whales, dolphins and turtles. Haines said his group is sensitive to those concerns and will take steps to minimize harm to marine animals, including stopping work when animals are seen nearby. The plan still needs to be approved by the National Oceanic and Atmospheric Administration.

http://www.wired.com/2014/07/cre-fivefold/

Resistant "Nightmare Bacteria" Increase Five-Fold in Southeastern U.S.

Cases of CRE rose five times over between 2008 and 2012

- **By Maryn McKenna**



***Klebsiella*, Janice Carr, CDC**

There's worrisome news here in the southeastern US, buried in a journal that is favorite reading only for superbug geeks like me. The rate at which hospitals are recognizing cases of CRE - the form of antibiotic resistance that is so serious the CDC dubbed it a "nightmare" - rose five times over between 2008 and 2012.

Within that bad news, there are two especially troubling points. First, the hospitals where this resistance factor was identified were what is called "community" hospitals, that is, not academic referral centers. That's an important distinction, because academic medical centers tend to be where the most cutting-edge care is performed, and where the sickest people are. As a result, they are where last-resort antibiotics are used the most, and therefore where resistance is most likely to emerge. That CRE was found so widely not in academic centers, but rather in community hospitals, is a signal that it is probably moving through what medicine calls "the community," which is to say, anywhere outside healthcare. Or, you know, everyday life.

A second concern is that the authors of the study, which is in *Infection Control and Hospital Epidemiology*, assume that their finding is an underestimate of the actual problem.

A little background first on CRE. (Archive of posts on it is here.) The acronym stands for "carbapenem-resistant *Enterobacteriaceae*." Enterobacteriaceae are a large family of bacteria that normally are carted around in your guts without causing illness. When they escape, though - for instance, during ICU treatment - they are a common cause of serious hospital-acquired infections. "Carbapenems" are a small group of very powerful antibiotics that are viewed as drugs of last resort, which work against infections that have become resistant to most other antibiotics. The acronym CRE indicates a group of resistant organisms that go by other acronyms - NDM, OXA, VIM and KPC, for instance - and that have been spreading across the globe for more than 10 years.

CREs are serious stuff: On average, at least half of those who contract CRE infections die. There are only a few antibiotics - sometimes one, sometimes two, depending on the organism - that work against them at all, and those drugs have significant problems and side effects. Broadly speaking, the emergence of CREs brings us several steps closer to the end of the antibiotic era.

For reasons that no one has ever been able to explain, one of the CRE organisms - KPC, or *Klebsiella pneumoniae* resistant to carbapenems - seems to have emerged in North Carolina; it was first noted in a set of bacterial samples that a hospital in that state sent to the CDC in 1996. So it's resonant that this study was conducted by researchers in North Carolina; it reveals how far that organism and others have spread.

About the study: It relies on data tendered to the Duke Infection Control Outreach Network by 25 community hospitals in North Carolina, South Carolina, Virginia and Georgia. The hospitals ranged in size from 100 to 657 beds, so some of them were truly small community institutions. The data was collected between January 2008 and December 2012, so as a snapshot of what is happened in the US with regard to CRE, it is pretty timely.

Out of the 25 hospitals, 16 identified 305 patients carrying or infected with CRE:

- ***59 percent had identifiable infections; 41 percent were colonized, that is, carrying the bacteria asymptomatically.***
- ***34 percent of the cases became evident while the patient was in the hospital (hospital-onset healthcare associated) and 60 percent after patients had returned home (community-onset hospital-associated)***
- ***of the cases that were diagnosed after someone had left an acute-care hospital, 56 percent were associated with nursing homes.***

The key trend is here: In 2008, the rate of CRE detection was 0.26 cases per 100,000 patient days; in 2012, it was 1.4 per 100,000 patient-days.

Those may seem like small numbers. Here is what the authors say:

…rates of CRE, while still infrequent, are increasing dramatically in community hospitals, where the majority of Americans receive their healthcare. We believe this increase is attributable to growing reservoirs and transmission of CRE and improvement in detection. Overall, we believe the estimates from study hospitals are underestimates of the true incidence in these hospitals. This point underscores the fact that these organisms are increasingly important and relevant in all areas of healthcare, including small community hospitals.

The study is worth reading as well for an extended discussion of the challenges of CRE detection, including the pace at which new laboratory standards for detecting these organisms are being adopted (or not). Overall, though, it is a worrisome indicator that highly resistant organisms may be outpacing our ability to detect or to treat them.

Cite: *Thaden JT, Lewis SS, Hazen KC et al. Rising Rates of Carbapenem-Resistant Enterobacteriaceae in Community Hospitals: A Mixed-Methods Review of Epidemiology and Microbiology Practices in a Network of Community Hospitals in the Southeastern United States. Infection Control and Hospital Epidemiology, Vol. 35, No. 8 (August 2014), pp. 978-983. DOI: 10.1086/677157*

http://www.bbc.com/news/health-28400968

Six seconds of exercise 'can transform health'

Short six-second bursts of vigorous exercise have the potential to transform the health of elderly people, say researchers in Scotland.

By James Gallagher Health editor, BBC News website

A pilot study involving 12 pensioners showed going all-out in very short bursts, reduced blood pressure and improved general fitness over time. The team at Abertay University believe it could help avert the "astronomical" costs of ill health in elderly people. Experts said the study emphasised the benefits of exercise at any age.

High Intensity Training (HIT) has attracted a lot of attention for promising some of the same benefits as conventional exercise but in a much shorter time. Instead of a comfortable half-hour jog or a few miles on the bike, HIT involves pushing yourself to your limits for a short period of time. The team in Scotland say they were conducting the first trials in older people.

Get a sweat on

A group of pensioners came into the lab twice a week for six weeks and went hell for leather on an exercise bike for six seconds. They would allow their heart rate to recover and then go for it again, eventually building up to one minute of exercise by the end of the trial. "They were not exceptionally fast, but for someone of that age they were," researcher Dr John Babraj said.

The results, published in the Journal of the American Geriatrics Society, showed participants had reduced their blood pressure by 9%, increased their ability to get oxygen to their muscles and found day-to-day activities like getting out of a chair or walking the dog easier.

Dr Babraj told the BBC the benefits could be huge: "We've got an ageing population and if we don't encourage them to be active, the economic burden of that is going to be astronomical. "A lot of diseases are associated with sedentary behaviour - like cardiovascular disease and diabetes - but if we can keep people active and functioning then we can reduce the risk. "Also on the social side, they are less likely to be socially active and will interact with people more."

More than 10 million people in the UK are over 65 and that figure is set to rise. Dr Babraj says older people struggle to exercise as many are full-time carers, but argues High Intensity Training would be easier to fit in. He said people could try it at home, but should see their doctor first to ensure there were no underlying health issues. "Then the easiest way to do it yourself is to run up a hill, the steeper the hill, the harder it's going to be, give it everything you've got for six seconds."

Safe?

There is an argument that short and strenuous exercise may be safer than conventional exercise. A higher heart rate and blood pressure caused by exercise can be a trigger for heart attacks and stroke. Dr Babraj said running for a long time "puts a greater strain on the heart overall" even if it is worked harder in the short-term in High Intensity Training. Larger trials are now planned.

Dr Adam Gordon, a consultant and honorary secretary of the British Geriatrics Society, told the BBC: "This is a brilliant, fantastic piece of work challenging assumptions about what the right type of exercise is in old age, but I'd encourage them to investigate the benefits in even older and even more frail people. "The broad message is that you're never too old, too frail, too ill to benefit from exercise, as long as it's carefully chosen. "We know even into your 80s and 90s there's a benefit from developing a very slight sweat by exercising on multiple occasions per week."

http://www.eurekalert.org/pub_releases/2014-07/cru-ndt072514.php

New drug target can break down cancer's barrier against treatment

Targeting a molecule in blood vessels can make cancer therapy significantly more effective

CANCER RESEARCH UK scientists at Barts Cancer Institute have found that targeting a molecule in blood vessels can make cancer therapy significantly more effective, according to research published in Nature today (Sunday).

The team at Barts Cancer Institute, part of Queen Mary University of London, have found that a molecule, called focal adhesion kinase (FAK), signals the body to repair itself after chemotherapy or radiotherapy, which kill cancer cells by damaging DNA. When the researchers removed FAK from blood vessels that grew in melanoma or lung cancer models, both chemotherapy and radiation therapies were far more effective in killing the tumours.

The researchers also studied samples taken from lymphoma patients. Those with low levels of FAK in their blood vessels were more likely to have complete remission following treatment. This suggests that developing drugs to strike out FAK in cancer blood vessels may boost cancer treatments and prevent cancer from coming back.

Dr Bernardo Tavora, lead author on the paper from the Barts Cancer Institute, said: "This work shows that sensitivity to cancer treatment is related to our own

body mistakenly trying to shield the cancer from cell-killing effects caused by radiotherapy and chemotherapy.

"Although taking out FAK from blood vessels won't destroy the cancer by itself, it can remove the barrier cancer uses to protect itself from treatment."

Cells lining the blood vessels send chemical signals, called cytokines, to the tumour to help it resist DNA damage and to recover. The researchers demonstrated that this process requires FAK in order to work, and without it, these signals are never sent – making the tumour more vulnerable to DNA damaging therapy.

Dr Kat Arney, Cancer Research UK's science communications manager, said: "This exciting research may have cracked how healthy cells in the blood vessels are protecting against cancer treatments. This research was only done in mice, but it gives real hope that we can boost the effectiveness of cancer medicine and sensitise cancers to the drugs we have."

** Tavora et al. Endothelial-FAK targeting sensitises tumours to DNA-damaging therapy. Nature 2014. DOI: 10.1038/nature13541*

http://bit.ly/1rsSeuF

US Doctor Infected With Ebola in Liberia Outbreak

An American doctor battling West Africa's Ebola epidemic has himself fallen sick with the disease in Liberia, his aid agency said.

Samaritan's Purse, a Christian charity, said Dr Kent Brantly had been isolated at the group's Ebola treatment center at the ELWA hospital in the Liberian capital Monrovia.

"Dr. Brantly is married with two children," the group said, in a statement posted to its website on Saturday.

"Samaritan's Purse is committed to doing everything possible to help Dr. Brantly during this time of crisis. We ask everyone to please pray for him and his family."

Brantly is the medical director of the Samaritan's Purse Ebola case management center in Liberia, where the agency continues to work with Liberian and international health officials to contain the outbreak.

Ebola is an hemorrhagic fever with a very high fatality rate. Liberia, Sierra Leone and Guinea have borne the brunt of the recent epidemic, and last week Nigeria recorded its first death.

As of July 20, the number of Ebola cases recorded in the months-long epidemic stood at 1,093, including more than 660 deaths, according to the World Health Organization.

The virus can fell victims within days, causing severe fever and muscle pain, vomiting, diarrhoea and, in some cases, organ failure and unstoppable bleeding.

Ebola is believed to be carried by animals hunted for meat, notably bats. It spreads among humans via bodily fluids including sweat, meaning you can get sick from touching an infected person.

With no vaccine, patients believed to have caught the virus must be isolated to prevent further contagion. Ebola first emerged in 1976 in what is now the Democratic Republic of Congo, and is named after a river there.