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Comet dust: Planet Mercury's 'invisible paint'

New research suggests that carbon from cometary material that bombards the Mercury may be the reason the planet's surface is so dark

PROVIDENCE, R.I. [Brown University] - A team of scientists has a new explanation for the planet Mercury's dark, barely reflective surface. In a paper published in Nature Geoscience, the researchers suggest that a steady dusting of carbon from passing comets has slowly painted Mercury black over billions of years.

Mercury's dark surface has long been a mystery to scientists. On average, Mercury is much darker than its closest airless neighbor, our Moon. Airless bodies are known to be darkened by micrometeorite impacts and bombardment of solar wind, processes that create a thin coating of dark iron nanoparticles on the surface. But spectral data from Mercury suggests its surface contains very little nanophase iron, certainly not enough to account for its dim appearance.

"It's long been hypothesized that there's a mystery darkening agent that's contributing to Mercury's low reflectance," said Megan Bruck Syal, a postdoctoral researcher at Lawrence Livermore National Laboratory who performed this research while a graduate student at Brown University. "One thing that hadn't been considered was that Mercury gets dumped on by a lot of material derived from comets."

As comets approach Mercury's neighborhood near the sun, they often start to break apart. Cometary dust is composed of as much as 25 percent carbon by weight, so Mercury would be exposed to a steady bombardment of carbon from these crumbling comets. Using a model of impact delivery and a known estimate of micrometeorite flux at Mercury, Bruck Syal was able to estimate how often cometary material would impact Mercury, how much carbon would stick to Mercury's surface, and how much would be thrown back into space. Her calculations suggest that after billions of years of bombardment, Mercury's surface should be anywhere from 3 to 6 percent carbon.

The next part of the work was to find out how much darkening could be expected from all that impacting carbon. For that, the researchers turned to the NASA Ames Vertical Gun Range. The 14-foot canon simulates celestial impacts by firing projectiles at up to 16,000 miles per hour.

For this study, the team launched projectiles in the presence of sugar, a complex organic compound that mimics the organics in comet material. The heat of an impact burns the sugar up, releasing carbon. Projectiles were fired into a material that mimics lunar basalt, the rock that makes up the dark patches on the nearside of the Moon. "We used the lunar basalt model because we wanted to start with something dark already and see if we could darken it further," said Peter Schultz, professor emeritus of geological sciences at Brown and a co-author of the new research.

The experiments showed that tiny carbon particles become deeply embedded in the impact melted material. The process reduced the amount of light reflected by the target material to less than 5 percent - about the same as the darkest parts of Mercury.

Importantly, spectroscopic analysis of the impact samples revealed no distinctive spectral fingerprints, again similar to flat spectral signatures from Mercury. "We show that carbon acts like a stealth darkening agent," Schultz said. "From the standpoint of spectral analysis, it's like an invisible paint."

And that paint has been building up on Mercury's surface for billions of years.

"We think this is a scenario that needs to be considered," Schultz said. "It appears that Mercury may well be a painted planet."

The research was supported by NASA's Planetary Geology and Geophysics program (NNX13AB75G) and the NASA Earth and Space Science Fellowship program (NNXC12AL79H). Miriam Riner from the Planetary Sciences Institute was a co-author on the paper.

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Glyburide associated with more risk of adverse events than insulin in newborns

Glyburid, used to treat gestational diabetes in pregnant women associated with higher risk for newborns to be admitted to a neonatal intensive care unit

The medication glyburide, which has been increasingly used to treat gestational diabetes in pregnant women, was associated with higher risk for newborns to be admitted to a neonatal intensive care unit, have respiratory distress, hypoglycemia (low blood glucose), birth injury and be large for gestational age compared with infants born to women treated with insulin, according to an article published online by JAMA Pediatrics.

The prevalence of gestational diabetes mellitus (GDM) in the United States has more than doubled during the last 20 years. Given the widespread and rapid use of glyburide in the last decade more evaluation of the comparative safety and effectiveness of the drug is needed. Previous literature on the association between treatment with glyburide and adverse neonatal outcomes is limited, according to background in the study.

Wendy Camelo Castillo, Ph.D., of the University of Maryland, Baltimore, and Michele Jonsson Funk, Ph.D., of the University of North Carolina at Chapel Hill, and coauthors estimated the risk of adverse maternal and neonatal outcomes in women with GDM treated with glyburide vs. insulin using data from a nationwide employer-based insurance claims database from 2000 through 2011. The authors

excluded women with type 1 or 2 diabetes as well as those younger than 15 and older than 45.

Among 110,879 women with GDM, 9,173 women (8.3 percent) were treated with glyburide (4,982 women) or insulin (4,191 women). Use of glyburide rose and the proportion of the group treated with glyburide increased from 8.5 percent in 2000 to 64.4 percent in 2011.

The authors found that among newborns whose mothers were treated with glyburide there was a 41 percent higher risk of neonatal intensive care unit admission, 63 percent higher risk of respiratory distress, 40 percent higher risk of hypoglycemia (low blood glucose), 35 percent higher risk of birth injury and 43 percent higher risk of being large for gestational age compared with newborns of women treated with insulin.

The difference in risk per 100 women associated with glyburide compared with insulin was 2.97 percent for neonatal intensive care unit admission, 1.41 percent for large for gestational age and 1.1 percent for respiratory distress.

Women treated with glyburide, as compared with insulin, were not at increased risk for obstetric trauma, preterm birth or jaundice. The risk of cesarean delivery was 3 percent lower in the glyburide group, according to the results.

"Given the widespread use of glyburide, further investigation of these differences in pregnancy outcomes is a public health priority," the study concludes.

(JAMA Pediatr. Published online March 30, 2015. doi:10.1001/jamapediatrics.2015.74. Available pre-embargo to the media at http://media.jamanetwork.com.)

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Editorial: Glyburide for Gestational Diabetes, Time for a Pause for Thought

In a related editorial, Richard I.G. Holt, Ph.D., F.R.C.P., of the University of Southampton, England, writes: "The major limitation with the current evidence has been the lack of power to demonstrate differences between insulin and glyburide, and this is particularly relevant for rare adverse events. The article by Camelo Castillo et al in this issue of JAMA Pediatrics is therefore a welcome addition to the debate."

"The main limitation of this and other observational analyses is that the results may be affected by important confounding factors. While the authors have adjusted for important medical conditions, they have not adjusted for all relevant sociodemographic features," Holt continues.

"This latest study heightens residual concerns about the use of glyburide to treat GDM that need to be resolved before this drug should be recommended for continued use in pregnancy. As the authors rightly conclude, the "higher risk of neonatal outcomes associated with glyburide-treated women demands further attention" and more attention is needed to determine which women are most likely to benefit from glyburide or perhaps more importantly not be harmed. It is time for a pause for thought," Holt concludes.

(JAMA Pediatr. Published online March 30, 2015. doi:10.1001/jamapediatrics.2015.144. Available pre-embargo to the media at http://media.jamanetwork.com.)

Editor's Note: The author made a conflict of interest disclosure. Please see article for additional information, including other authors, author contributions and affiliations, etc.

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Short bouts of high-intensity exercise before a fatty meal best for vascular health

Short burst of intensive exercise before eating a high fat meal is better for blood vessel function in young people

A short burst of intensive exercise before eating a high fat meal is better for blood vessel function in young people than the currently recommended moderate-intensity exercise, according to a new study from the University of Exeter.

Cardiovascular diseases including heart attacks and stroke are the leading cause of death in the UK, and the process underlying these diseases start in youth. An impairment in the function of blood vessels is thought to be the earliest event in this process, and this is known to occur in the hours after consuming a high fat meal.

Performing exercise before a high fat meal is known to prevent this impairment in blood vessel function, but no study has yet identified what type of exercise is best.

The study, published in the American Journal of Physiology - Heart and Circulatory Physiology, compared high-intensity, interval exercise against moderate-intensity exercise on blood vessel function in adolescent boys and girls after they had consumed a high fat milkshake.

It showed that approximately 25 minutes of moderate-intensity cycling prevented the fall in blood vessel function after the high fat meal. However, performing just eight minutes of high-intensity cycling not only prevented this fall, but improved blood vessel function to a level that was superior to moderate-intensity exercise.

Dr Alan Barker, of the Children's Health and Exercise Research Centre, Sport and Health Sciences at the University of Exeter, said: "Our study shows that the intensity of exercise plays an important part in protecting blood vessel function in young people after the ingestion of a high fat meal."

"Furthermore, both the boys and girls found the high-intensity exercise to be more enjoyable than the moderate-intensity exercise. Considering that very few adolescents currently achieve the recommended minimum of one hour of at least moderate-intensity exercise per day, smaller amounts of exercise performed at a

higher-intensity might offer an attractive alternative to improve blood vessel function in adolescents." The researchers say the next step is to move the work beyond healthy adolescents and study those with risk factors for cardiovascular disease, such as obesity and type I diabetes.

'Exercise intensity and the protection from postprandial vascular dysfunction in adolescents' by B. Bond, P.E Gates, S.R Jackman, L.M Corless, C.A Williams and A.R Barker is published in the American Journal of Physiology - Heart and Circulatory Physiology.

http://www.eurekalert.org/pub_releases/2015-03/sfhe-elt033015.php

Endoscopes linked to outbreak of drug-resistant E. coli

An outbreak of a novel Escherichia coli (E. coli) strain resistant to antibiotics has been linked to contaminated endoscopes in a Washington state hospital.

NEW YORK - The study indicates that industry standard cleaning guidelines, which were exceeded by hospital staff, may not be sufficient for sterilizing endoscopes adequately. The research was published online in Infection Control & Hospital Epidemiology, the journal of the Society for Healthcare Epidemiology of America. "Although the endoscopes had been reprocessed according to industry standards, we identified contaminated endoscopes that might have facilitated the transmission of the multidrug-resistant organism," said Kristen Wendorf, MD, MS, lead author of the study. "In the wake of the recent outbreak of CRE due to contaminated endoscopes, we suspect endoscope-associated transmission of bacteria is more common than recognized and not adequately prevented by current reprocessing guidelines."

During a period of November 2012-August 2013, a hospital in Washington state experienced an outbreak of the rare E. coli bacteria, initially identified through molecular testing of isolate bacteria by the Washington State Public Health Laboratory. Testing identified a cluster of carbapenem-resistant E. coli with distinct genetic markers, suggesting a common source.

Researchers collaborated with hospital staff to conduct a public health investigation to determine the extent of the outbreak, identify potential sources of transmission and design, and implement infection control measures to prevent future cases.

The investigation identified 32 patients with the specific bacteria. All patients had severe pancreatic or biliary disease and had undergone endoscopic retrograde cholangiopancreatography (ERCP). A manufacturer review of the endoscope cleaning procedures found the hospital's process to be above industry standards. However, the review found serious defects in the endoscopes that were not apparent during hospital testing. While testing the scopes for bacteria, researcher also found half of the reprocessed scopes harbored bacteria, including the two used in ERCP procedures that tested positive for the specific E. coli bacteria. Even after an overhaul by the manufacturer, these scopes harbored bacteria in the elevator channel.

More than 30 percent of patients infected with the bacteria died during the investigation and seven of the deaths occurred during hospitalization within 30 days of the date the E. coli isolate was obtained, although it is not possible to determine whether an infection contributed to the deaths. The primary diagnoses for the patients who died included pancreatic cancer, colon cancer, primary sclerosing cholangitis, and renal/pancreatic transplant.

"The outbreak was detected through a public health surveillance program that was enhanced with the addition of molecular testing, and would likely have gone undetected otherwise," said Wendorf. "Routine surveillance is crucial for promptly recognizing outbreaks and monitoring and responding to the ongoing threat from multidrug-resistant organisms in healthcare facilities."

As a result of this outbreak, the hospital has undertaken costly and extraordinary measures to minimize risk for endoscope-related infection transmission. The facility now quarantines ERCP scopes after cleaning and does not release them for use until cultures are negative at 48 hours. Despite these additional safeguards, the hospital's scopes continue to show signs of bacteria after cleaning and require additional cleaning before the next use.

While there are industry standards for cleaning these devices, maintenance guidelines are not available from the manufacturers. The researchers note the need to include evaluation and maintenance schedules in the approval processes of these devices in moving forward to ensure adequate cleaning processes.

Kristen Wendorf, Meagan Kay, Christopher Baliga, Scott Weissman, Michael Gluck, Punam Verma, Maria D'Angeli, Jennifer Swoveland, Mi-Gyeong Kang, Kaye Eckmann, Andrew Ross, Jeffrey Duchin. "Endoscopic Retrograde Cholangiopancreatography-Associated AmpC Escherichia coli." Infection Control & Hospital Epidemiology. Web. (March XX, 2015).

http://www.eurekalert.org/pub_releases/2015-03/asfn-cop033015.php

Consumption of peanuts with a meal benefits vascular health

A study of peanut consumption showed that including them as a part of a high fat meal improved the post-meal triglyceride response and preserved endothelial function.

"Peanuts are a healthy snack when eaten as part of a healthy diet," said lead researcher Xiaoran Liu, a graduate student in the Department of Nutritional Sciences at The Pennsylvania State University.

The purpose of this research was to evaluate vascular function after a high fat meal challenge. Overweight males (n = 15) were randomized to either a peanut meal containing 3 oz. of ground peanuts (as a shake) or a control meal (a shake without peanuts) that were matched for energy and macronutrients.

The lipid profile, glucose and insulin were measured five times after each meal. Flow-mediated dilatation (FMD) was measured to assess vascular function. This non-invasive method required a cuff at the forearm to restrain blood flow, which was then released to assess dilation of the brachial artery. The control meal decreased FMD by 1.2% compared to baseline. In contrast, there was no decrease in FMD after the peanut meal. These results demonstrate that the peanut meal maintained normal vascular function whereas the high fat-matched control meal impaired vascular function acutely. Vascular dysfunction plays a major role in the development of atherosclerosis and the formation of coronary plaques and lesions that lead to coronary artery disease. Typically after a high fat meal, vascular function is reduced, albeit temporarily, until the fat that is in the blood (from the meal) is cleared. Strategies that can blunt this response to both dietary fat and its effect on vascular dysfunction may decrease the risk of coronary disease. Our finding demonstrated that that peanut consumption was shown to be atheroprotective as a part of high fat meal.

"Previous studies have shown that individuals who consume peanuts more than 2 times a week have a lower risk of coronary heart disease," said Liu. "This study indicates that the protective effect of peanut consumption could be due, in part, to its beneficial effect on artery health".

Peanuts are nutrient dense and energy dense, so Liu noted the importance of being aware of their calorie content when incorporating them in the diet. Thus, peanuts must replace other food sources of calories when included in the diet. For example, peanuts can be substituted for high fat, nutrient-poor foods in the diet that contain solid fats.

Looking ahead, the Penn State group hopes to investigate the effects of peanut consumption on other risk factors including inflammatory markers. Liu presented the research at the American Society for Nutrition's Scientific Sessions & Annual Meeting at EB 2015. *The study was supported by The Peanut Institute.*

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Electroconvulsive therapy changes key areas of the human brain that play a role in memory, emotion

Findings may help physicians to pre-select for patients who will respond to treatment

Although scientists know that depression affects the brain, they don't know why some people respond to treatment while others do not.

Now a team of UCLA researchers has shown for the first time in a large cohort of patients that electroconvulsive therapy (ECT), sometimes referred to as shock treatment, change certain areas of the brain that play a role in how people feel, learn and respond to positive and negative environmental factors. The team imaged the hippocampus and amgydalar in patients before, during and after undergoing ECT and compared those images to healthy control subjects.

They also showed that the hippocampus changes, or increases in size, correlated to improved mood in patients with major depression and indicated how well they were responding to treatment. Additionally, using leading-edge methods to look at brain shape, the team showed that parts of these structures change more with treatment, providing vital clues to how the connections in the brain may be used to select for patients who will respond well to treatment.

That would also result in sparing those patients who won't respond from months of taking drugs that ultimately won't work for them, said study senior author Katherine L. Narr, an associate professor of neurology.

"Major depression is common, affects all ages, races and ethnic groups and has a serious consequence on people's family lives and work," Narr said. "People with depression also are at higher risk for suicide, which accounts for more deaths than car accidents, natural disasters and war each year on average. Unfortunately, standard types of medication used to treat major depression take a long time to work, and for at least a third of people, the medication will not work well enough to provide any real help." The study appears in the early online edition of the peer-reviewed journal Biological Psychiatry.

ECT, which has been used for more than 50 years, carries with it a certain stigma. However, within the last decade, advances in anesthesia, electrical stimulation equipment and new evidence about electrode lead placement have improved safety and reduced side effects, said study first author Shantanu H. Joshi, an assistant professor of neurology.

Further advances in high-resolution MRI also allow the measurement of the induced brain changes with improved accuracy and precision.

"ECT has been shown to be very effective for treating patients with major depression who don't respond well to other treatments," Joshi said. "During the treatment course, ECT leads to plastic changes in the brain that are linked with improvements in mood. Specifically, we saw the hippocampus and amygdala - important for memory and emotion - are shown to increase in size. People with smaller hippocampal size prior to starting treatment are less likely to respond as well to treatment. While our research investigates structural neuroplasticity in depression in response to ECT, our findings are considered to be of much broader interest to the field."

In addition to ECT, the team expects that the effects shown would extend to more standard, less rapidly acting antidepressant treatments and could be used to predict patient response.

In this study, the team imaged 43 patients undergoing ECT at three time points, before beginning treatment, after the second ECT session and within one week of completing treatment, resulting in 129 brain scans. They also imaged 32 healthy controls twice, and compared those images to the ECT patients.

Going forward, the UCLA team will examine the relationship between the hippocampal structural neuroplasticity and its neurochemistry in terms of the metabolite response, which has important implications for understanding the brain's metabolic regulation and the excitation and suppression as a response to ECT. Additionally, the hippocampal and amgydalar shape will be used as features for classification and prediction of depression diagnosis and treatment response using advanced machine-learning techniques. The changes in the hippocampal and the amgydalar structure will be further investigated with regards to novel disease maintenance treatments along with relapse/recurrence rates.

Major depression affects 350 million people each year and leads to enormous personal suffering, loss of productivity and is aburden to family, the health care system and the economy. Finding better ways to select patients for treatments that will alleviate their symptoms would go a long way to reducing that suffering, Narr said.

"Our findings newly show that hippocampal structure prior to ECT may be an important indicator of treatment outcome," the study states. "That is, patients with smaller hippocampal volumes at baseline are shown to more likely exhibit increases in volume with ECT and to show concomitant improvements in clinical symptoms. Results further indicate that both clinical response to ECT and ECT-induced changes in volume occur rapidly."

The study was funded by the National Institute of Mental Health (RO1MH092301 and K2MH102743).

http://www.eurekalert.org/pub_releases/2015-03/uoc - slu032615.php

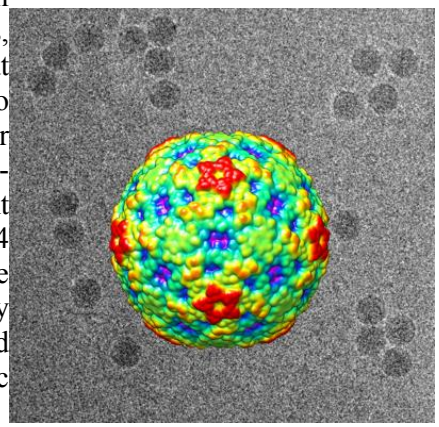
Scientists link unexplained childhood paralysis to enterovirus D68

UCSF-led team rules out other pathogens with comprehensive sequencing

A research team led by UC San Francisco scientists has found the genetic signature of enterovirus D68 (EV-D68) in half of California and Colorado children diagnosed with acute flaccid myelitis - sudden, unexplained muscle weakness and paralysis - between 2012 and 2014, with most cases occurring during a nationwide outbreak of severe respiratory illness from EV-D68 last fall.

The finding strengthens the association between EV-D68 infection and acute flaccid myelitis, which developed in only a small fraction of those who got sick. The scientists could not find any other pathogen capable of causing these symptoms, even after checking patient cerebrospinal fluid for every known infectious agent.

Researchers analyzed the genetic sequences of EV-D68 in children with acute flaccid myelitis and discovered that they all corresponded to a new strain of the virus, designated strain B1, which emerged about four years ago and had mutations similar to those found in poliovirus and another closely related nerve-damaging virus, EV-D70. The B1 strain was the predominant circulating strain detected during the 2014 EV-D68 respiratory outbreak, and the researchers found it both in respiratory secretions and - for the first time - in a blood sample from one child as his acute paralytic illness was worsening.



This is a three-dimensional image of enterovirus D68 (center) reconstructed from cryo-electron micrographs (background). **Yue Liu and Michael Rossmann, Purdue University.**

The study also included a pair of siblings, both of whom were infected with genetically identical EV-D68 virus, yet only one of whom developed acute flaccid myelitis.

"This suggests that it's not only the virus, but also patients' individual biology that determines what disease they may present with," said Charles Chiu, MD, PhD, an associate professor of Laboratory Medicine and director of UCSF-Abbott Viral Diagnostics and Discovery Center. "Given that none of the children have fully recovered, we urgently need to continue investigating this new strain of EV-D68 and its potential to cause acute flaccid myelitis."

Among the 25 patients with acute flaccid myelitis in the study, 16 were from California and nine were from Colorado. Eleven were part of geographic clusters of children in Los Angeles and in Aurora, Colorado, who became symptomatic at the same time, and EV-D68 was detected in seven of these patients.

Although the researchers found EV-D68 in the children's respiratory secretions and in the blood from one case, they did not find it in cerebrospinal fluid. The researchers said this may not be surprising given that other nerve-damaging viruses, like polio, are very rarely detected in cerebrospinal fluid.

Eighty percent of the children reported having an upper respiratory illness about six days, on average, before their acute flaccid myelitis symptoms began. Slightly more reported having a fever, including all of the cases from the clusters in California and Colorado.

Samples were collected more than a week after the children began showing symptoms of an upper respiratory infection, and this likely made it much harder to

find EV-D68. There may also be other reasons to explain why the virus was not found in cerebrospinal fluid in children with neurological symptoms.

"The lack of detectable virus in CSF could also mean that the neurological symptoms are coming from an aberrant immune response to recent EV-D68 infection and not because the virus is directly invading neurons," Chiu said.

This study was supported by grants from the National Institutes of Health, a University of California Discovery Award, an Abbott Viral Discovery Award and the Centers for Disease Control and Prevention Emerging Infections Program.

Other authors include Alexander Greninger, MD, PhD, Samia Naccache, PhD, Guixia Yu, BS, Sneha Somasekar, BS, Scot Federman, BA and Doug Stryke, BS, of UCSF; Kevin Messacar, MD and Samuel Dominguez, MD, PhD, of Children's Hospital Colorado and University of Colorado School of Medicine, Aurora; Anna Clayton, BS, MPH, Christopher Anderson, BS, Shigeo Yagi, PhD, Sharon Messenger, PhD, Debra Wadford, PhD, Dongxiang Xia, MD, PhD, and Carol Glaser, DVM, MD, of the California Department of Public Health; Keith Van Haren, MD, of Lucile Packard Children's Hospital at Stanford University; and Grace Aldrovandi, MD, of Children's Hospital Los Angeles and University of Southern California.

http://bit.ly/1IummOs

Anglo-Saxon remedy kills hospital superbug MRSA

Thousand-year-old Anglo-Saxon recipe to vanquish a stye also kills MRSA

by Clare Wilson

Take cropleek and garlic, of both equal quantities, pound them well together… take wine and bullocks gall, mix with the leek… let it stand nine days in the brass vessel… So goes a thousand-year-old Anglo-Saxon recipe to vanquish a stye, an infected eyelash follicle. The medieval medics might have been on to something. A modern-day recreation of this remedy seems to alleviate infections caused by the bacteria that are usually responsible for styes. The work might ultimately help create drugs for hard-to-treat skin infections.

The project was born when Freya Harrison, a microbiologist at the University of Nottingham, UK, got talking to Christina Lee, an Anglo-Saxon scholar. They decided to test a recipe from an Old English medical compendium called Bald's Leechbook, housed in the British Library. Some of the ingredients, such as copper from the brass vessel, kill bacteria grown in a dish – but it was unknown if they would work on a real infection or how they would combine.

Careful collection

Sourcing authentic ingredients was a major challenge, says Harrison. They had to hope for the best with the leeks and garlic because modern crop varieties are likely to be quite different to ancient ones – even those branded as heritage. For the wine they used an organic vintage from a historic English vineyard.

As "brass vessels" would be hard to sterilise – and expensive – they used glass bottles with squares of brass sheet immersed in the mixture. Bullocks gall was easy, though, as cow's bile salts are sold as a supplement for people who have had their gall bladders removed. After nine days of stewing, the potion had killed all the soil bacteria introduced by the leek and garlic. "It was self-sterilising," says Harrison. "That was the first inkling that this crazy idea just might have some use."

A side effect was that it made the lab smell of garlic. "It was not unpleasant," says Harrison. "It's all edible stuff. Everyone thought we were making lunch."

The potion was tested on scraps of skin taken from mice infected with methicillin-resistant Staphylococcus aureus. This is an antibiotic-resistant version of the bacteria that causes styes, more commonly known as the hospital superbug MRSA. The potion killed 90 per cent of the bacteria. Vancomycin, the antibiotic generally used for MRSA, killed about the same proportion when it was added to the skin scraps.

A loathsome slime

Unexpectedly, the ingredients had little effect unless they were all brought together. "The big challenge is trying to find out why that combination works," says Steve Diggle, another of the researchers. Do the components work in synergy or do they trigger the formation of new potent compounds?

Using exactly the right method also seems to be crucial, says Harrison, as another group tried to recreate the remedy in 2005 and found that their potion failed to kill bacteria grown in a dish. "With the nine-day waiting period, the preparation turned into a kind of loathsome, odorous slime," says Michael Drout of Wheaton College in Norton, Massachusetts. If the 9th Century recipe does lead to new drugs, they might be useful against MRSA skin infections such as those that cause foot ulcers in people with diabetes. "These are usually antibiotic-resistant," says Diggle. However, he doesn't recommend people try this at home.

It wouldn't be the first modern drug to be derived from ancient manuscripts – the widely used antimalarial drug artemisinin was discovered by scouring historical Chinese medical texts. Harrison is due to present the research at the Society for General Microbiology conference in Birmingham, UK, this week.

http://bit.ly/1ChloA0

Opossum Compounds Isolated to Help Make Antivenom

And researchers have engineered a common bacteria to inexpensively create the snakebite treatment

By Matt Davenport and Chemical & Engineering News

A simple peptide could save countless future snakebite victims in developing countries, researchers announced at the American Chemical Society national meeting in Denver. The antivenom relies on a sequence of just 11 amino acids, copied from an opossum protein.

The research team, led by Claire F. Komives of San Jose State University, also demonstrated that genetically modified bacteria could produce the protective peptide at low costs.

Komives unveiled the antivenom candidate in a Division of Biochemical Technology session in Denver on Sunday. But the fundamental discoveries behind the findings were made nearly 20 years ago by a researcher named Binie V. Lipps. Opposums have an innate immunity to a variety of snake venoms. Lipps isolated the protein responsible for this immunity and found that peptides containing its first 10 or 15 amino acids seemed to contain all of the protein's antivenomous properties. She first patented the work in 1996.

It was widely ignored until 2012, when online news outlets stumbled on the opossum protein thanks to a blogger writing about the remarkable survival ability of opossums. Komives learned about the protein from Yahoo! News while searching for ideas for a research project that would allow her to work with a colleague in India while on sabbatical.

Although deaths from snakebites are incredibly rare in the U.S., they are surprisingly common in India. According to some estimates, snakes are responsible for 100,000 deaths in the country every year, Komives said, and rural areas, where snakebites are most common, don't always have access to antivenoms.

Komives decided to try and develop a more inexpensive method for producing the natural opossum product and applied for a Fulbright scholarship.

Working with collaborators at the Indian Institute of Technology Delhi, Komives engineered Escherichia coli to synthesize peptides containing multiple repeats of the first 11 amino acids of the opossum protein. The researchers then use a protease to cleave at the final amino acid in that sequence to release the individual peptides.

Producing the peptides simply required ordering a plasmid and growing engineered bacteria, Komives said, but the team does need to optimize the peptide purification process. She estimates that once scaled up, companies could produce the peptides at roughly $1.00 per g.

Meanwhile, collaborators at the National Natural Toxins Research Center at Texas A&M University, Kingsville, have confirmed that synthetic versions of the 11-mer peptide protect mice against the hemorrhagic effects of venom from Russell's viper (Daboia russelii), a common snake in India, and help mice survive exposure to rattlesnake venom.

Michael G. Thomas, a bacteriologist at the University of Wisconsin, Madison, and a chair of the session in Denver, said he was impressed by how much of an impact this simple peptide could make.

"The bottom line is the peptide clearly does something," said Komives, who is working to establish a crowd-sourcing campaign to better understand its efficacy. "Somebody needs to be working on this."

http://bit.ly/1ChloA0

It Doesn't Matter How Much Time Parents Spend With Their Kids

New research shows no link between amount of time spent with children and emotional, behavioral, or academic outcomes

By Erin Blakemore smithsonian.com

American mothers spend more time with their kids today than they did in the 1960s, partly due to assumptions that the more time parents and kids spend together, the better. But new research could turn that assumption on its head in a big way - as the Washington Post reports, a new study shows that the amount of time parents spend with kids has "virtually no relationship to how children turn out."

For the first time ever, researchers have undertaken a large longitudinal study of how parents spend their time and how their kids perform, Brigid Schulte reports. The study used time diaries and survey data to track how accessible mothers were to their children, linking that data to kids' outcomes in the areas of behavior, emotion and academics. It found that the amount of time spent with kids "did not matter" - and in some cases could even harm children.

Schulte explains that time spent with stressed mothers can actually hurt children. Guilty, anxious moms who struggle to juggle work and childcare were linked to worse outcomes like lower math scores and behavioral problems. But overall, the study's authors found that time spent with mothers doesn't really matter - except during adolescence, when an engaged mom can result in less delinquent behavior.

The study flies in the face of the notion that a mother's one-on-one time with her child is "sacred." But the study's authors note that the quality of time spent with kids still matters, even though the results don't point to a magic number of time kids should be spending with their parents. In fact, notes Schulte, there's another factor that predicted success more reliably than any amount of time spent with kids - social resources like "income and a mother's educational level."

"In an ideal world, this study would alleviate parents' guilt about the amount of time they spend, and show instead what's really important for kids," Melissa Milkie, who co-authored the study, told Schulte. In the meantime, other research is pointing to the potential downsides of overly involved parents - this Wall Street Journal report points to a study that suggests that helicopter parents may increase their kids' risk of physical inactivity.

http://www.eurekalert.org/pub_releases/2015-03/uot-had033015.php

How a deadly fungus evades the immune system

New research from the University of Toronto has scientists re-thinking how a lethal fungus grows and kills immune cells.

The study hints at a new approach to therapy for Candida albicans, one of the most common causes of bloodstream infections.

Previously, scientists thought that Candida albicans spread by changing from a single, round cell to a long string of cells, or filaments. They thought this shape change allowed the fungus to move through the bloodstream and let its filaments penetrate tissues and destroy immune cells.

But the new study, published today in Nature Communications, shows that a little bit of sugar on the surface of fungal cells triggers the death of immune cells that would otherwise kill the fungus.

"It's not the shape-change per se that enables the fungus to kill the immune cell, but what happens along with it," says Professor Leah Cowen, lead researcher on the study who holds the Canada Research Chair in Microbial Genomics and Infectious Disease in U of T's Department of Molecular Genetics. "The addition of glycosylated proteins, which are proteins with a sugar attached, re-models the surface of the fungal cells."

Cowen and her lab found that Candida albicans can kill immune cells even after its cells have died. They let immune cells called macrophages consume the fungus, and after an hour they removed the fungal cells from the macrophages. Then they exposed new macrophages to fungal cells that had been consumed and those that had not, and they compared the results.

"The fungal cells that were never internalized by macrophages couldn't kill the fresh macrophages, but those that had been inside a macrophage could kill beautifully," says Cowen. That finding was a clue. The researchers reasoned that the change in the fungal cells that turned them into killers was probably on their surface, since dead cells have no active internal processes.

The researchers then used an enzyme called Endo H to snip off sugars on the glycosylated proteins attached to the dead fungal cells. The change completely blocked the ability of the fungus to kill - a strong lead on a new and needed therapeutic strategy for Candida albicans.

Globally, fungi kill more than 1.5 million people a year. In the U.S., Candida fungi account for almost 90 per cent of hospital-acquired fungal infections, and in Canada they're the third most common cause of bloodstream infections in intensive care units. More than 40 per cent of people with a systemic Candida albicans infection will die.

A therapy that targets the ability of fungal cells to outfox the immune system would be promising, says Cowen, because it might minimize effects on healthy microbes and avoid spurring drug resistance.

As well, some anti-fungals in development - including one in Cowen's lab - are hindered because the target proteins are present in both fungi and humans. That means a drug has to distinguish between the fungal and human versions of the target. "If you develop a drug that targets something that's only found in fungi, it's less likely to have side effects in a human," says Cowen.

In her Nature Communications study, Cowen used a powerful Candida albicans mutant library, which the pharmaceutical company Merck recently made public. The library let Cowen and her team test the function of almost all genes in the Candida albicans genome, where before they could test just 10 per cent. "It really let us approach this pathogen from a holistic perspective and evaluate the role of all its genes in disease," says Cowen.

The researchers used the library to do the first genome-scale analysis of the fungus's ability to change shape and grow, and they discovered more than 800 regulators of this process, which they published today with their other findings.

"It's cool because we have a ton of new biology to explore, hundreds of possible drug targets and a new appreciation of how fungal pathogens interact with immune systems," says Cowen. "It's been a lot of fun."

http://www.eurekalert.org/pub_releases/2015-03/sri-sru033015.php

Scientists reveal unique mechanism of natural product with powerful antimicrobial action

Unique mechanism of a powerful natural product with wide-ranging antifungal, antibacterial, anti-malaria and anti-cancer effects is revealed

JUPITER, FL - Scientists from the Florida campus of The Scripps Research Institute (TSRI) have uncovered the unique mechanism of a powerful natural product with wide-ranging antifungal, antibacterial, anti-malaria and anti-cancer effects.

The new study, published online ahead of print by the journal Nature Communications, sheds light on the natural small molecule known as borrelidin.

"Our study may help the rational design of compounds similar to borrelidin with a range of useful applications, particularly in cancer," said Min Guo, a TSRI associate professor who led the study.

Powerful Medicines

Guo and his colleagues were interested in borrelidin, because it inhibits a specific type of enzyme known as threonyl-tRNA synthetase (ThrRS), ultimately impeding protein synthesis.

Compounds similar to borrelidin have been used as treatments for microbial infections. For example, the natural product mupirocin is approved as a topical treatment for bacterial skin infections and febrifugine (the active component of the Chinese herb Chang Shan (Dichroa febrifuga Lour)) has been used for treating malaria-induced fever for nearly 2,000 years.

Previous studies from the collaborator Professor Christopher S. Francklyn of the University of Vermont College of Medicine and others have shown that borrelidin impedes angiogenesis, the growth of new blood vessels critical for the spread of malignant tumors, as well as increasing apoptosis in certain types of leukemia.

"It is probably the most potent tRNA synthetase inhibitor on Earth, "said Research Associate Pengfei Fang, co-first author of the study and member of the Guo lab at Scripps Florida. "It is also the earliest known tRNA synthetase inhibitor, discovered in 1966 - just a few years after people learned the existence of tRNA synthetase and genetic code."

Research Associate Xue Yu, also co-first author of the study and a member of the Guo lab, emphasized, "While little is known about how borrelidin works, the fairly widespread use of these types of inhibitors highlights their tremendous potential in a number of medical applications."

Winning at Musical Chairs

In the new study, the scientists set out to conduct a detailed structural and functional analysis of the binding of borrelidin to both human and bacterial (E. coli) ThrRS in the hope of identifying its unique mechanism.

The researchers succeeded, and the new study shows for the first time that borrelidin occupies four distinct subsites on both the bacterial and human tRNA synthetase, including all three subsites for its normal binding substrates and an extra one that is created when the compound binds. In this way, borrelidin crowds out all natural partners that would otherwise bind those sites and fuel the process of protein synthesis.

In that sense, borrelidin more or less wins the game of molecular musical chairs by taking over everyone's seat well before the music starts, even including the aisles.

Because each of the subsites is essential for its activity, the fact that borrelidin occupies four subsites within ThrRS, an apparent inhibitory overkill, was a quite surprise, and indeed accounts for its potency as validated by further experiments done in both in vitro and in cells.

"This has never been seen in any other tRNA synthetase inhibitors, including the ones sold as medicines," said Guo. "This finding establishes a new inhibitor class and highlights the striking design of this natural compound that inhibits tRNA synthetases in two of the three kingdoms of life."

In addition to Guo, Fang and Yu, other authors of the study, "Structural Basis for Full-Spectrum Inhibition of Translational Functions on a tRNA Synthetase," are Kaige Chen and Xin Chen of TSRI; Seung Jae Jeong and Sunghoon Kim of Seoul National University, Korea; and Adam Mirando and Christopher S. Francklyn the University of Vermont College of Medicine.

The work was supported by the National Institutes of Health (grants NIEHS T32 ES007122-23, GM54899, GM100136 and GM106134), the Korean Global Frontier Project (NRF-M1AXA002-2010-0029785), and the PGA Women's Cancer Awareness Foundation.

http://www.eurekalert.org/pub_releases/2015-03/uos-wfs033115.php

World first study reveals antibodies that may trigger psychosis in children

A world first study revealing the presence of two antibodies in a sub-group of children experiencing their first episode of psychosis affirms a longstanding recognition that auto-immune disorders play a significant role in psychiatric illness.

Antibodies defend the body against bacterial, viral, and other invaders but sometimes the body makes antibodies that attack healthy cells. In these cases, autoimmune disorders develop. These include conditions such as multiple sclerosis (MS), rheumatoid arthritis and Type 1 diabetes. This 'immune hypothesis' is supported by new work colleagues in the current issue of Biological Psychiatry

Researchers from the Kids Research Institute at the Children's Hospital, Westmead, and the University of Sydney detected antibodies to the dopamine D2 receptor or the N-methyl-D-aspartate (NMDA) glutamate receptor among eight out of 43 children experiencing their first episode of psychosis, but no such antibodies in healthy children.

Both are key neural signaling proteins previously been implicated in psychosis.

"The antibodies we have detected in children having a first episode of acute psychosis suggest there is a distinct subgroup for whom autoimmunity plays a role in their illness," says the University of Sydney's Dr Fabienne Brilot, the senior author on the paper and Head of the Neuroimmunology Group at The Children's Hospital at Westmead in Sydney.

"The finding suggests that better interventions are possible, providing hope that major disability can be prevented for the subset of children experiencing acute psychosis with antibodies," Brilot adds.

Dopamine is a chemical messenger aiding the transmission of signals in the brain and other areas of the body. Regulating its actions plays a crucial role in mental and physical health. Dopamine acts on receptors tailored specifically for it. The dopamine-2 receptor (D2R) is one of five subtypes of mammalian dopamine.

Increasing knowledge of the roles of dopamine receptor subtypes raises the hope that more selective drugs will be developed. Abnormalities in dopaminergic neurotransmission play a key role in the pathogenesis of psychosis. Many drugs affect dopamine transmission directly by either blocking or stimulating its receptors. Many antipsychotics show varying affinities for the different dopamine receptors but blockade of the dopamine-2 receptor (D2R) specifically has proved to be indispensable in the clinical management of psychosis.

While less well established than dopamine, it is also likely that glutamatergic dysfunction also plays a role in psychotic disease. This suggests that specific pathologies and processes affecting D2R and the glutamatergic N-methyl-D-aspartate receptor (NMDAR) could define biological subgroups and may be involved in the pathogenesis of psychosis and other psychiatric illnesses such as schizophrenia.

"There is a pressing need in psychiatry to establish biologically based disease subtypes, which might allow for more specific diagnosis and effective intervention," says Dr Brilot. "Our findings contribute further understanding of the biology of psychiatric and neurological diseases and whether autoantibodies detected in a subgroup of patients can trigger psychiatric disorders.

"Further research will reveal whether these antibodies are the mark of a clinically relevant subset of patients and, if so, whether immunosuppressive therapies can effectively treat children with these debilitating illnesses."

http://www.eurekalert.org/pub_releases/2015-03/apa-isc033115.php

Internet searches create illusion of personal knowledge, research finds

Inflated sense of personal knowledge may have negative effects, study concludes

WASHINGTON - Searching the Internet for information may make people feel smarter than they actually are, according to new research published by the American Psychological Association.

"The Internet is such a powerful environment, where you can enter any question, and you basically have access to the world's knowledge at your fingertips," said lead researcher Matthew Fisher, a fourth-year doctoral candidate in psychology at Yale University. "It becomes easier to confuse your own knowledge with this external source. When people are truly on their own, they may be wildly inaccurate about how much they know and how dependent they are on the Internet."

In a series of experiments, participants who searched for information on the Internet believed they were more knowledgeable than a control group about topics unrelated to the online searches. In a result that surprised the researchers, participants had an inflated sense of their own knowledge after searching the Internet even when they couldn't find the information they were looking for. After conducting Internet searches, participants also believed their brains were more active than the control group did. The research was published online in the Journal of Experimental Psychology: General.

For nine experiments, a range of 152 to 302 participants were recruited online, with different participants taking part in each experiment. In one experiment, the Internet group used online searches to research four questions (e.g., "How does a zipper work?") and provided a website link with the best answer. The control group was given the exact text from the most common website used by the Internet group to answer the questions. Both groups then rated their ability to answer other questions (e.g., "Why are cloudy nights warmer?") on topics unrelated to the Internet searches, although they didn't have to answer those questions. The Internet group members consistently rated themselves as more knowledgeable than the control group about those unrelated topics.

The Internet group reported an inflated sense of personal knowledge after Internet searches even when its members could not find complete answers to very difficult questions (e.g., "Why is ancient Kushite history more peaceful than Greek history?") or when they found no answers at all because of Google filters that were used. The cognitive effects of "being in search mode" on the Internet may be so powerful that people still feel smarter even when their online searches reveal nothing, said study co-author Frank Keil, PhD, a psychology professor at Yale.

In another experiment, participants who did online searches thought their brains would be more active than the control group, and they chose magnetic resonance images of a brain with more active areas highlighted as representative of their own brains. This result suggests that the participants searching the Internet believed they had more knowledge in their heads, rather than simply thinking they knew more because they had access to the Internet, Fisher said.

The use of Internet searches, not just access to the Internet, appeared to inflate participants' sense of personal knowledge. When the Internet group members were given a particular website link to answer questions, they didn't report higher levels of personal knowledge on the unrelated topics than the control group.

People must be actively engaged in research when they read a book or talk to an expert rather than searching the Internet, Fisher said. "If you don't know the answer to a question, it's very apparent to you that you don't know, and it takes time and effort to find the answer," he said. "With the Internet, the lines become blurry between what you know and what you think you know."

The growing use of smartphones may exacerbate this problem because an Internet search is always within reach, Keil said, and the effects may be more pronounced when children who are immersed in the Internet from an early age grow up to be adults.

An inflated sense of personal knowledge also could be dangerous in the political realm or other areas involving high-stakes decisions, Fisher said.

"In cases where decisions have big consequences, it could be important for people to distinguish their own knowledge and not assume they know something when they actually don't," he said. "The Internet is an enormous benefit in countless ways, but there may be some tradeoffs that aren't immediately obvious and this may be one of them. Accurate personal knowledge is difficult to achieve, and the Internet may be making that task even harder."

Article: "Searching for Explanations: How the Internet Inflates Estimates of Internal Knowledge;" Matthew Fisher, MA, Mariel K. Goddu, BA, and Frank C. Keil, PhD; Yale University; Journal of Experimental Psychology: General, online, Mar. 31, 2015.

Full text of the article is available from the APA Public Affairs Office and at http://www.apa.org/pubs/journals/releases/xge-0000070.pdf.

http://www.eurekalert.org/pub_releases/2015-03/nsf-tn033115.php

The 'intraterrestrials': New viruses discovered in ocean depths

Viruses infect methane-eating archaea beneath the seafloor

The intraterrestrials, they might be called.

Strange creatures live in the deep sea, but few are odder than the viruses that inhabit deep ocean methane seeps and prey on single-celled microorganisms called archaea.

The least understood of life's three primary domains, archaea thrive in the most extreme environments on the planet: near hot ocean rift vents, in acid mine drainage, in the saltiest of evaporation ponds and in petroleum deposits deep underground.

Virus in the deep blue sea

While searching the ocean's depths for evidence of viruses, scientists have found a remarkable new one, a virus that seemingly infects archaea that live beneath the ocean floor. The researchers were surprised to discover that the virus selectively targets one of its own genes for mutation, and that this capacity is also shared by archaea themselves. The findings appear today in a paper in the journal Nature Communications.

The project was supported by a National Science Foundation (NSF) Dimensions of Biodiversity grant to characterize microbial diversity in methane seep ecosystems. Dimensions of Biodiversity is supported by NSF's Directorates for Biological Sciences and Geosciences.

New information about life in ocean depths

"Life far beneath the Earth's subsurface is an enigma," said Matt Kane, program director in NSF's Division of Environmental Biology. "By probing deep into our planet, these scientists have discovered new information about Earth's microbes and how they evolve."

"Our study uncovers mechanisms by which viruses and archaea can adapt in this hostile environment," said David Valentine, a geoscientist at the University of California Santa Barbara (UCSB) and co-author of the paper.

The results, he said, raise new questions about the evolution and interaction of the microbes that call the planet's interior home. "It's now thought that there's more biomass inside the Earth than anywhere else, just living very slowly in this dark, energy-limited environment," said paper co-author Sarah Bagby of UCSB.

Using the submersible Alvin, Valentine and colleagues collected samples from a deep-ocean methane seep by pushing tubes into the ocean floor and retrieving sediments. The contents were brought back to the lab and fed methane gas, helping the methane-eating archaea in the samples to grow.

When the team assayed the samples for viral infection, they discovered a new virus with a distinctive genetic fingerprint that suggested its likely host was methane-eating archaea.

Genetic sequence of new virus holds the key

The researchers used the genetic sequence of the new virus to chart other occurrences in global databases. "We found a partial genetic match from methane seeps off Norway and California," said lead author Blair Paul of UCSB. "The evidence suggests that this viral type is distributed around the globe in deep ocean methane seeps."

Further investigation revealed another unexpected finding: a small genetic element, known as a diversity-generating retroelement, that accelerates mutation of a specific section of the virus's genome. Such elements had been previously identified in bacteria and their viruses, but never among archaea or the viruses that infect them. "These researchers have shown that cutting-edge genomic approaches can help us understand how microbes function in remote and poorly known environments such as ocean depths," said David Garrison, program director in NSF's Division of Ocean Sciences.

While the self-guided mutation element in the archaea virus resembles known bacterial elements, the researchers found that it has a divergent evolutionary history. "The target of guided mutation - the tips of the virus that make first contact when infecting a cell - is similar," said Paul. "But the ability to mutate those tips is an offensive countermeasure against the cell's defenses, a move that resembles a molecular arms race."

Unusual genetic adaptations

Having found guided mutation in a virus-infecting archaea, the scientists reasoned that archaea themselves might use the same mechanism for genetic adaptation.

In an exhaustive search, they identified parallel features in the genomes of a subterranean group of archaea known as nanoarchaea.

Unlike the deep-ocean virus that uses guided mutation to alter a single gene, the nanoarchaea target at least four distinct genes. "It's a new record," said Bagby.

"Bacteria had been observed to target two genes with this mechanism. That may not seem like a huge difference, but targeting four is extraordinary."

According to Valentine, the genetic mutation that fosters these potential variations may be key to the survival of archaea beneath the Earth's surface.

"The cell is choosing to modify certain proteins," he said. "It's doing its own protein engineering. While we don't yet know what those proteins are being used for, learning about the process can tell us something about the environment in which these organisms thrive."

Viral DNA sequencing was provided through a Gordon and Betty Moore Foundation grant. The research team also included scientists from the University of California, Los Angeles; the University of California, San Diego; and the U.S. Department of Energy's Joint Genome

http://www.eurekalert.org/pub_releases/2015-03/mali-ccb033115.php

Can caffeine be used to treat or prevent Alzheimer's disease?

The proposed link between caffeine and reductions in the beta amyloid plaque accumulation characteristic of Alzheimer's disease (AD) suggest a possible role for caffeine in AD treatment.

New Rochelle, NY - The latest evidence linking beta amyloid protein to Alzheimer's disease and exploring the relationship between caffeine and beta amyloid are featured in a review article in Journal of Caffeine Research: The International Multidisciplinary Journal of Caffeine Science, a peer-reviewed journal from Mary Ann Liebert, Inc., publishers.

The article is available free on the Journal of Caffeine Research website at http://online.liebertpub.com/doi/full/10.1089/jcr.2014.0027 until May 1, 2015.

In the article "Caffeine as Treatment for Alzheimer's: A Review", Abhishek Mohan, BS, Old Dominion University (Norfolk, VA), and coauthors identify the potential opportunities for using caffeine to reduce beta amyloid levels as a means of preventing, treating, and slowing the progression of Alzheimer's disease.

"To say that strategizing medicines to treat Alzheimer's disorders is important is an understatement," says Patricia A. Broderick, PhD, Editor-in-Chief of Journal of Caffeine Research, Medical Professor in Physiology, Pharmacology & Neuroscience, The Sophie Davis School of Biomedical Education, The City College of New York, The City University of New York, and Adjunct Professor in Neurology, New York University Langone Medical Center and Comprehensive Epilepsy Center. "Moreover, to say that caffeine is just an ordinary staple in our lives, whether caffeine is part of coffee or a chocolate bar, is also an understatement. Thus, what Dr. Mohan has published herein is elegant in its simplicity; his work is critically on target."

http://www.eurekalert.org/pub_releases/2015-03/uoc - ri033115.php

Researchers identify 'beige' fat-burning cells in humans

Energy-burning cells hold potential for new anti-obesity drugs

For the first time, a research team, led by a UC San Francisco biologist, has isolated energy-burning "beige" fat from adult humans, which is known to be able to convert unhealthy white fat into healthy brown fat. The scientists also found new genetic markers of this beige fat.

The discovery is an important advance in the search for new medications to fight obesity, said senior investigator Shingo Kajimura, PhD, UCSF assistant professor of cell and tissue biology, with a joint appointment in the UCSF Diabetes Center and the Eli and Edythe Broad Center of Regeneration Medicine and Stem Cell Research at UCSF.

The finding was published online on March 16, 2015 in Nature Medicine.

All mammals, including humans, have two types of fat with completely opposite functions: white, which stores energy and is linked with diabetes and obesity, and brown, which produces heat by burning energy and is associated with leanness. Human babies are born with brown fat as a natural defense against cold, and hibernating animals such as bears build up large stores of brown fat for the same reason.

Since 2009, explained Kajimura, it's been known that adult humans also have significant amounts of brown fat. But until now, it had not been known whether this fat is the so-called classical brown fat of the type that babies are born with, or beige fat, which is found within white fat and has the ability to convert, or recruit, white fat into brown fat in response to cold or other stresses.

To answer that question, Kajimura and his team isolated and cloned single brown fat cells from two adult individuals. After sophisticated genetic and protein analyses of the cloned cells, they concluded that they had successfully isolated recruitable brown fat.

"This finding brings us another step closer to the goal of our laboratory, which is engineering fat cells to fight obesity," said Kajimura. "We are trying to learn how to convert white fat into brown fat, and until now, it had not been demonstrated that this recruitable form of brown fat is actually present in humans."

Now that they have a reliable human beige fat cell culture system, Kajimura said, his team will be able to use the system as a screening platform to identify and test

small molecules that activate the development, differentiation, and thermogenic (heat-producing) activity of human brown fat.

The ultimate aim, he said, is the creation of drugs to turn white fat into brown fat through brown fat recruitment.

"If you think about obesity, it's generally caused by an imbalance between energy intake and energy expenditure," Kajimura said. "So far, all of the approved anti-obesity medications reduce energy intake by decreasing appetite. They work in the short term, but they often have side effects such as depression. If we have a compound that increases energy expenditure by recruiting new brown fat and activating brown fat thermogenesis, then it might work synergistically with conventional anti-obesity medications. This would be a novel approach to modulating whole-body energy balance."

Co-authors of the study are Kosaku Shinoda, PhD, Ineke H. N. Luijten, Yutaka Hasegawa, MD, PhD, Haemin Hong, Si B. Sonne, PhD, and Miae Kim, PhD, of UCSF; Ruidan Xue PhD, of Joslin Diabetes Center (JBS) and Harvard Medical School (HMS); Maria Chondronikola of Shriners Hospital for Children (SHC) and University of Texas (UT); Aaron M Cypess, MD, PhD, and Yu-Hua Tseng, PhD, of JBS and HMS; Jan Nedergaard, PhD, of Stockholm University, Sweden; and Labros S. Sidossis, PhD, of SHC and UT.

The study was supported by funds from the National Institutes of Health, Pew Charitable Trust, Japan Science and Technology Agency, Japan Society for the Promotion of Science, and Manpei Suzuki Diabetes Foundation.

http://www.eurekalert.org/pub_releases/2015-03/uota-udc033115.php

UT Dallas criminologist challenges effectiveness of solitary confinement

Study finds that the punishment does not deter further violence in prison

A new study by a UT Dallas criminologist finds that solitary confinement does not deter inmates from committing further violence in prison.

Dr. Robert Morris, associate professor of criminology and director of the Center for Crime and Justice Studies in the School of Economic, Political and Policy Sciences, tracked the behavior of 3,808 male inmates in 70 Texas prisons. He compared general population inmates who received solitary confinement for an act of violent misconduct with those who did not receive the punishment for the same type of offense. Solitary confinement typically restricts inmates to their cells for 23 hours a day.

In January, the study was published ahead of print in the Journal of Quantitative Criminology.

The prisoners in the study who received solitary confinement were no more - or less - violent behind bars after the punishment, according to the study. Solitary confinement also did not affect how soon an inmate committed further violent acts while incarcerated.

Morris said he hopes the findings generate discussion about the effectiveness of solitary confinement in regular prison settings. The research did not focus on higher security prisons that more extensively use solitary confinement.

"You're not getting a reward one way or the other for exposing inmates to solitary, so you have question its utility," Morris said. "It's costing money, it's costing time and there are potentially harmful side effects."

The study cites previous research that has found that solitary confinement can cause serious health and psychological problems for inmates, many of whom are vulnerable because of existing mental health conditions and/or addictions.

Reducing the use of solitary confinement also may save taxpayer funds, Morris said. Solitary confinement may be necessary for temporary periods to break up violent situations, but that based on the research, it should be used with caution, he said. Texas law prohibits solitary confinement for more than 15 consecutive days.

"A lot of people may argue that it's a necessary tool in the prison," Morris said. "Its administration could probably be improved because there's so much discretion involved, and there's so little known about what exposure can lead to."

Morris said more research is needed on how solitary confinement affects prisoners once they are released.

"The vast majority of these folks will return to society, so you don't want to aggravate their prison experience any more than you have to. If you're aggravating circumstances inside, then it could be that you're aggravating circumstances when they come out," he said. "Then, you're raising the chance you might see them again, and at that point, you're just wasting tax dollars."

http://www.eurekalert.org/pub_releases/2015-03/yu-iam033115.php

In Alzheimer's mice, memory restored with cancer drug

Memory and as well as connections between brain cells were restored in mice with a model of Alzheimer's given an experimental cancer drug, Yale School of Medicine researchers reported in the journal Annals of Neurology.

The drug, AZD05030, developed by Astra Zeneca proved disappointing in treating solid tumors but appears to block damage triggered during the formation of amyloid-beta plaques, a hallmark of Alzheimer's disease. The new study, funded by an innovative National Institutes of Health (NIH) program to test failed drugs on different diseases, has led to the launch of human trials to test the efficacy of AZD05030 in Alzheimer's patients.

"With this treatment, cells under bombardment by beta amyloid plaques show restored synaptic connections and reduced inflammation, and the animal's memory, which was lost during the course of the disease, comes back," said

Stephen M. Strittmatter, the Vincent Coates Professor of Neurology and senior author of the study.

In the last five years, scientists have developed a more complete understanding of the complex chain of events that leads to Alzheimer's disease.

The new drug blocks one of those molecular steps, activation of the enzyme FYN, which leads to the loss of synaptic connections between brain cells. Several other steps in the disease process have the potential to be targets for new drugs, Strittmatter said.

"The speed with which this compound moved to human trials validates our New Therapeutic Uses program model and serves our mission to deliver more treatments to more patients more quickly," said Christopher P. Austin, M.D., director of NIH's National Center for Advancing Translational Sciences (NCATS), which funded the work.

Yale's Christopher H. van Dyck, a co-author of the paper, and Strittmatter have initiated a multi-site clinical trial to determine whether the drug can also benefit Alzheimer's patients. For more information, visit https://clinicaltrials.gov/ (NCT02167256 and NCT01864655).

The study was funded by the NCATS and the NIH Common Fund, through the Office of Strategic Coordination/Office of the NIH Director

http://www.eurekalert.org/pub_releases/2015-03/miot-trr033115.php

The rapid rise of human language

New paper suggests people quickly started speaking in a now-familiar form

At some point, probably 50,000 to 100,000 years ago, humans began talking to one another in a uniquely complex form. It is easy to imagine this epochal change as cavemen grunting, or hunter-gatherers mumbling and pointing.

But in a new paper, an MIT linguist contends that human language likely developed quite rapidly into a sophisticated system: Instead of mumbles and grunts, people deployed syntax and structures resembling the ones we use today.

"The hierarchical complexity found in present-day language is likely to have been present in human language since its emergence," says Shigeru Miyagawa, Professor of Linguistics and the Kochi Prefecture-John Manjiro Professor in Japanese Language and Culture at MIT, and a co-author of the new paper on the subject.

To be clear, this is not a universally accepted claim: Many scholars believe that humans first started using a kind of "proto-language" - a rudimentary, primitive kind of communication with only a gradual development of words and syntax.

But Miyagawa thinks this is not the case. Single words, he believes, bear traces of syntax showing that they must be descended from an older, syntax-laden system, rather than from simple, primal utterances.

"Since we can find syntax within words, there is no reason to consider them as 'linguistic fossils' of a prior, presyntax stage," Miyagawa adds.

Miyagawa has an alternate hypothesis about what created human language: Humans alone, as he has asserted in papers published in recent years, have combined an "expressive" layer of language, as seen in birdsong, with a "lexical" layer, as seen in monkeys who utter isolated sounds with real-world meaning, such as alarm calls.

Miyagawa's "integration hypothesis" holds that whatever first caused them, these layers of language blended quickly and successfully.

Word to the wise

Miyagawa's paper is published this month in the peer-reviewed journal Frontiers in Psychology. Vitor A. Nobrega of the University of Sao Paulo co-authored the paper.

In the paper, Nobrega and Miyagawa write that a single word can be "internally complex, often as complex as an entire phrase," making it less likely that words we use today are descended from a presyntax mode of speech.

To see a straightforward example of this in English, take "nationalization," Miyagawa suggests. It starts with "nation," a noun; adds "-al" to create an adjective; adds "-iz(a)" to form a verb; and ends with "-tion," to form another noun, albeit with a new meaning.

"Hierarchical structure is present not only in single words, but also in compounds, which, contrary to the claims of some, are not the structureless fossilized form of a prior stage," Miyagawa says.

In their paper, Nobrega and Miyagawa hold that the same analysis applies to words in Romance languages that have been described elsewhere as remnants of formless proto-languages.

In Brazilian Portuguese, "porta asciuga-mani" - literally "carry dry-hands," but today colloquially meaning "towel holder" - is one such case, they contend, where a compound derived from old words has a clear internal structure. (In this case, "dry hands" is a complement to the verb.)

Miyagawa's integration hypothesis is connected intellectually to the work of other MIT scholars, such as Noam Chomsky, who have contended that human languages are universally connected and derive from our capacity for using syntax. In forming, this school of thought holds, languages have blended expressive and lexical layers through a system Chomsky has called "Merge."

"Once Merge has applied integrating these two layers, we have essentially all the features of a full-fledged human language," Miyagawa says.

http://bit.ly/1GRtF4l

Spending Too Much Time on Homework Linked to Lower Test Scores

A new study suggests the benefits to homework peak at an hour a day. After that, test scores decline.

By Samantha Larson smithsonian.com

Polls show that American public high school teachers assign their students an average of 3.5 hours of homework a day. According to a recent study from the University of Oviedo in Spain, that's far too much.

While doing some homework does indeed lead to higher test performance, the researchers found the benefits to hitting the books peak at about an hour a day.

In surveying the homework habits of 7,725 adolescents, this study suggests that for students who average more than 100 minutes a day on homework, test scores start to decline. The relationship between spending time on homework and scoring well on a test is not linear, but curved.

This study builds upon previous research that suggests spending too much time on homework leads to higher stress, health problems and even social alienation. Which, paradoxically, means the most studious of students are in fact engaging in behavior that is counterproductive to doing well in school.

Because the adolescents surveyed in the new study were only tested once, the researchers point out that their results only indicate the correlation between test scores and homework, not necessarily causation. Co-author Javier Suarez-Alvarez thinks the most important findings have less to do with the amount of homework than with how that homework is done. From Education Week:

Students who did homework more frequently – i.e., every day – tended to do better on the test than those who did it less frequently, the researchers found. And even more important was how much help students received on their homework – those who did it on their own preformed better than those who had parental involvement. (The study controlled for factors such as gender and socioeconomic status.)

"Once individual effort and autonomous working is considered, the time spent [on homework] becomes irrelevant," Suarez-Alvarez says. After they get their daily hour of homework in, maybe students should just throw the rest of it to the dog.

http://www.eurekalert.org/pub_releases/2015-04/uoc - poh040115.php

Presence of heart pouch may explain strokes of unknown origin, UCI study finds

Anatomical variant could promote stagnation of blood, forming clots that migrate

Irvine, Calif - A pouchlike structure inside the heart's left atrial chamber in some people may explain strokes that otherwise lack an identifiable cause, according to UC Irvine School of Medicine researchers. Dr. Mark Fisher, a professor of neurology and pathology & laboratory medicine, and colleagues evaluated 75 stroke patients at UC Irvine Medical Center to learn whether this left atrial septal pouch could be a potent source of stroke-causing blood clots. Of the 23 patients who had experienced a stroke of undetermined origin (a "cryptogenic" stroke), 30 percent possessed the left atrial septal pouch. It was present in only 10 percent of the 52 patients who'd had a stroke with an identifiable trigger.

Stroke is the leading cause of long-term severe disability and the fourth-most-common cause of death in the U.S. About 80 percent of the 700,000-plus strokes that occur annually in this country are due to blood clots blocking a brain artery. In up to a third of these cases, the clots' origin cannot be determined. UC Irvine cardiologists first discovered this pouchlike structure inside the heart's left atrial chamber in a 2010 study.

"The cul-de-sac nature of this heart pouch may promote stagnation of the blood, forming clots that can travel into the brain and cause a stroke," Fisher said. "This finding points to a potentially important cause of strokes," he added. "The presence of this pouch could change how neurologists treat these patients and lead to new therapeutic strategies for preventing strokes."

Fisher said that large-scale studies are necessary to verify the results of this study, which appears online in Frontiers in Neurology at http://journal.frontiersin.org/article/10.3389/fneur.2015.00057/abstract.

The research was conducted at UC Irvine Medical Center by members of the Department of Neurology (Fisher and Dr. Annlia Paganini-Hill), the Division of Cardiology (Drs. Dawn Lombardo, Nathan Wong, Ailin Barseghian, Jashdeep Dhoot, Harkawal Hundal and Jonathan Salcedo) and the UCI School of Medicine (Dr. Jonathan Wong, who is now with the California Pacific Medical Center). It was supported by the American Heart Association.

http://www.eurekalert.org/pub_releases/2015-04/nsf-rie040115.php

Researchers improve efficiency of human walking

Unpowered exoskeleton developed by Carnegie Mellon and North Carolina State researchers helps individuals walk using less energy

Humans have evolved to be incredibly efficient at walking. In fact, simulations of human locomotion show that walking on level ground and at a steady speed should theoretically require no power input at all.

But anyone who works on their feet or has taken an arduous hike knows otherwise. In fact, people expend more energy during walking than any other activity in daily life, and for the elderly and those with mobility issues, that energy can be precious. For decades, engineers have envisioned systems that could make walking easier. In fact, so many researchers have tried to build unpowered exoskeletons and

failed that it was hotly debated in the field whether it was even possible to improve the efficiency of walking without adding an external energy source.

In news reported today in Nature, researchers from Carnegie Mellon University and North Carolina State University have demonstrated an unpowered ankle exoskeleton that reduces the metabolic cost of walking by approximately 7 percent. The results are roughly the equivalent of taking off a 10-pound backpack, and are equivalent to savings from exoskeletons that use electrically-powered devices. The research was based upon work supported by the National Science Foundation.

This image shows walking with a passive-elastic ankle exoskeleton. An unpowered clutch engages a spring in parallel with the Achilles tendon when the foot is on the ground, offloading the calf muscles and making walking easier. **Stephen Thrift, North Carolina State University**

"It's a real exciting milestone for the field of assistive devices," said Thomas Roberts, a professor of ecology and evolutionary biology at Brown University and an expert in the biomechanics of locomotion, who was not involved in the research. "They've taken an assistive device and lowered the cost of human walking. That's kind of a big deal because walking is already really cheap, and they did it with a very simple, but clever device."

The device is the result of eight years of patient and incremental work, mapped out on a whiteboard by Steve Collins and Greg Sawicki when they were graduate students together at the University of Michigan in 2007.

"Walking is more complicated than you might think," said Collins, an assistant professor of mechanical engineering at Carnegie Mellon. "Everyone knows how to walk, but you don't actually know how you walk."

Collins, Sawicki and co-author M. Bruce Wiggin succeeded where so many in the past had failed by performing careful analyses of the biomechanics of human walking and then designing a simple, ultra-light-weight device that relieved the calf muscle of its efforts when it wasn't doing any productive work.

Ultrasound imaging studies had revealed that the calf muscle exerts energy not only when propelling the body forward, but also when it performs a clutch-like action, holding the Achilles tendon taut.

"Studies show that the calf muscles are primarily producing force isometrically, without doing any work, during the stance phase of walking, but still using substantial metabolic energy," Collins explained. "This is the opposite of regenerative braking. It's as if every time you push on the brake pedal in your car, you burn a little bit of gas."

With this insight in mind, the team created an ankle exoskeleton that offloads some of the clutching muscle forces of the calf, reducing the overall metabolic rate.

A mechanical clutch engages when the foot is on the ground and disengages when the foot is in the air, to avoid interfering with toe clearance. This clutch takes over the effort of the calf, producing force without using consuming any energy and thereby reducing the overall metabolic rate.

In developing the device, the research team faced a challenge. When you place heavy objects on the legs, there's an initial penalty that increases your energy costs. Previous efforts had not been able to overcome that initial penalty. For that reason, it was critical to the researchers to keep the device light.

Over several years and many iterative designs, the team developed a carbon-fiber design that is ultra-light, yet rugged and functional. The entire device weighs approximately one pound per leg, or less than a work boot.

According to experts, the device is a triumph of elegance, simplicity and bio-specific interventions over complex, over-engineered designs.

"This unexpected and unprecedented result, with the potential to improve such a familiar human activity as walking, was discovered during a fundamental scientific study of mechanically augmented ankle function," said Jordan Berg, a program director at NSF. "It is a great example of how basic research can lead to new beneficial devices."

One of the long-term goals of Collins and Sawicki's project is to use lightweight, energy-efficient exoskeletons to assist individuals with mobility issues.

"You can imagine these lightweight efficient devices being worn on the affected limb to help people with the permanent aftereffects of stroke," Collins said. "We're hopeful that designs that use similar techniques can help people who have had a stroke walk more easily. We're still a little ways away from doing that, but we certainly plan to try."

In the future, the team intends to test the current device with individuals who have a variety of mobility issues to determine what designs might work best for different populations. They are also interested in developing exoskeleton components for the knee and the hip, where they believe they may be able to garner even larger benefits.

"As we understand human biomechanics better, we've begun to see wearable robotic devices that can restore or enhance human motor performance," said Collins. "This bodes well for a future with devices that are lightweight, energy-efficient, and relatively inexpensive, yet enhance human mobility."

http://www.eurekalert.org/pub_releases/2015-04/jhm-sal040115.php

Study affirms lethal prostate cancer can spread from other metastatic sites

Original tumor not the only agent of proliferation

A new genomic analysis of tissue from patients with prostate cancer has added more evidence that cells within metastases from such tumors can migrate to other body parts and form new sites of spread on their own.

Results of the analysis undermine anew long-held beliefs that cells with metastatic potential originate solely from the original or primary site of a cancer, according to the scientists who performed the study.

"The idea that metastatic tumors can seed and establish other metastatic tumors in patients is different from traditional theories that the primary tumor is solely responsible for disseminating cancer cells with metastatic potential," says William Isaacs, Ph.D., the William Thomas Gerrard, Mario Anthony Duhon and Jennifer and John Chalsty Professor of Urology at the Johns Hopkins Brady Urological Institute and a member of The Johns Hopkins Kimmel Cancer Center. "The new genomic information lends more support to the idea that treatments for metastatic cancers should be a combination of therapies that target a variety of genetic pathways."

Data used in the analysis, described in a report of the work online April 1 in Nature, were generated from a novel set of samples, collected in a Johns Hopkins autopsy program for patients with prostate cancer from 1995 through 2004. The new work comprised extensive genome sequencing and bioinformatics analysis of tumor samples by scientists at the Wellcome Trust Sanger Institute, University of Tampere in Finland and members of the International Cancer Genome Consortium, who found that the genetic makeup of cells within metastatic prostate tumors matched the makeup of new tumors from other metastatic sites.

Specifically, the investigators used a catalog of the genetic code of 51 tumors removed from 10 men who died from prostate cancer and were autopsied at The Johns Hopkins Hospital, as well as a sample of normal tissue from each of them.

Whole-genome sequencing on the samples showed that "even though a single cell begins the metastatic process, the disease becomes very heterogeneous as it spreads throughout the body over time, both between and among individuals. In individual patients, each metastatic site becomes an entity unto itself," says Isaacs, who also is a professor of oncology at the Johns Hopkins University School of Medicine.

The scientists found that five of the 10 men had patterns of mutations across several metastatic lesions, suggesting that these lesions were derived from not one but multiple metastatic sites. In seven of the men, the metastatic tumors were genetically more similar to each other than to the primary tumor.

The current findings expand on results of a Johns Hopkins-led study of autopsy samples published in Nature Medicine in 2009, conducted by scientist G. Steven Bova, M.D., who was then at Johns Hopkins and is now at the University of Tampere in Finland. That study showed similar patterns of genetic similarities across metastatic sites. The current study provides more detail and insight into the metastatic process, says Isaacs.

Isaacs says the current study relied on a novel set of tissue samples obtained from metastatic prostate cancer patients who, along with their families, agreed to be autopsied when they ultimately died from the disease. "Nearly every tissue and bone in the body was biopsied, and Dr. Bova and the autopsy team collected thousands of samples from these men who generously donated their tissues to science," says Isaacs.

Bova, Isaacs and their team at Johns Hopkins began the autopsy program in 1994 at a time when there was little access to metastatic prostate cancer tissue and when genome sequencing technology did not exist. "These samples, along with their annotations, are even more valuable now since we can use them in very sophisticated genetic studies such as the current one," says Isaacs. "The contributions these men made will hopefully produce more illuminating results that will pave the way for better treatment and prevention of prostate cancer."

Funding for the study was provided by Cancer Research UK, the Academy of Finland, the Cancer Society of Finland, the PELICAN Autopsy Study family members and friends from the Johns Hopkins Brady Urological Institute, John and Kathe Dyson, the National Institutes of Health's National Cancer Institute (CA92234), the American Cancer Society, the Johns Hopkins University Department of Pathology, the Women's Board of The Johns Hopkins Hospital, The Grove Foundation, the Association for the Cure of Cancer of the Prostate, the American Foundation for Urologic Disease, the Bob Champion Cancer Trust, the Research Foundation - Flanders (FWO), David Koch and the Prostate Cancer Foundation.

In addition to Bova, the study's leader, and Isaacs, the following scientists contributed to the research: Gunes Gundem, Peter Van Loo, Barbara Kremeyer, Ludmil B. Alexandrov, Jose M.C. Tubio, Elli Papaemmanuil, Victoria Goody, Calli Latimer, Sarah O'Meara, Kevin J. Dawson, Peter J. Campbell, Ultan McDermott and David C. Wedge from the Wellcome Trust Sanger Institute; Daniel S. Brewer from Norwich Medical School and the University of East Anglia in Norwich, U.K.; Heini M.L. Kallio, Gunilla Högnäs, Matti Annala, Kati Kivinummi, Matti Nykter and Tapio Visakorpi from the University of Tampere; Michael R. Emmert-Buck from the National Cancer Institute; Christopher Foster from the University of Liverpool and HCA Pathology Laboratories, London; Zsofia Kote-Jarai, Colin S. Cooper and Rosalind A. Eeles from the Institute of Cancer Research, London; Douglas Easton from the University of Cambridge in the U.K.; Hayley C. Whitaker and David E. Neal from Cancer Research UK; and the International Cancer Genome Consortium's Prostate Group.

http://www.eurekalert.org/pub_releases/2015-04/qub-qsd040115.php

Queen's scientists develop first perfume which smells better the more you sweat

First-ever perfume delivery system to ensure the more a person sweats, the better they will smell

The first-ever perfume delivery system to ensure the more a person sweats, the better they will smell, has been developed by scientists at Queen's University Belfast.

Researchers in the Queen's University Ionic Liquid Laboratories (QUILL) Research Centre have developed a unique new perfume delivery system which releases more of its aroma when it comes into contact with moisture, meaning a person smells nicer when their sweat levels increase.

This innovative perfume system has been created by tagging a raw fragrance onto an ionic liquid (salt in the form of liquid) which has no smell.

The 'perfumed ionic liquid' releases its aroma when it comes into contact with water, allowing more of the perfume's scent to be released onto a person's skin.

In addition, the perfume system also has the ability to remove the bad odours that come from sweat.

The 'thiol' compounds that are responsible for the malodour of sweat are attracted to the ionic liquid, attaching themselves to it and losing their potency.

The breakthrough could have major commercial possibilities, potentially providing a new way to develop products for the huge personal care market. QUILL researchers are currently working with a perfume development company to identify a number of product ideas that could eventually be sold in shops.

Project leader, Dr Nimal Gunaratne, from the Queen's University Belfast Ionic Liquid Laboratories (QUILL) Research Centre, said: "This is an exciting breakthrough that uses newly discovered ionic liquid systems to release material in a controlled manner. Not only does it have great commercial potential, and could be used in perfumes and cosmetic creams, but it could also be used in others area of science, such as the slow release of certain substances of interest."

"This innovative development demonstrates the drive of researchers at Queen's to advancing knowledge and achieving excellence for the benefit of society as a whole."

The research was carried out by corresponding author Dr Nimal Gunaratne, Professor Ken Seddon and Dr Peter Nockemann, from the Queen's University QUILL Research Centre.Read the full research article at:

http://pubs.rsc.org/en/content/articlelanding/2015/cc/c5cc00099h#!divAbstract

http://www.eurekalert.org/pub_releases/2015-04/foas-fso040115.php

Failed synchronization of the womb's clock with mother's body clock critical in miscarriages

New research in The FASEB Journal suggests that body clock genes are switched off in the lining of the womb to allow an embryo to implant and that regulation of this process is critical to successful pregnancy

If you are trying to have a baby, a good night's sleep is more important than ever. A new research report appearing in The FASEB Journal shows that the womb has its own "body clock" that needs to synchronize with the mother's body clock to ensure optimal conditions for fetal growth and development. The inability of a mother's body clock to synchronize with the womb's clock may be at least part of the reason why some women have difficulty carrying a pregnancy to full term. Specifically, the failed synchronization switches off body clock genes in cells lining the womb, which in turn, may jeopardize the pregnancy. This information may help researchers and fertility experts develop strategies to optimize the fetal environment to help more women have children.

"Infertility affects one in six couples across the world. Miscarriage is the most common complication of pregnancy," said Jan Brosens, M.D., a researcher involved in the work from the Division of Translational and Systems Medicine and Reproductive Health at Warwick Medical School at the University of Warwick in Coventry, UK. "Approximately one in seven clinical pregnancies result in miscarriage, mostly prior to 12 weeks of pregnancy. It is estimated that five percent of women experience two clinical miscarriages and approximately one percent have three or more losses. From a medical perspective, recurrent miscarriages and implantation failure have remained frustratingly devoid of effective therapeutic strategies."

To make this discovery, Brosens and colleagues, obtained womb biopsies from 70 women who have experienced recurrent pregnancy loss. The cells from these biopsies were purified and then treated in such a way as to simulate a pregnancy. They found that failure of embryonic and maternal body clock genes to synchronize could have catastrophic consequences. Not only did they find that this could cause miscarriage or infertility, but they also found more subtle synchronization defects could increase the risk of complications in the later stages of pregnancy, such as pre-eclampsia, fetal growth restriction and pre-term birth. This work also provides new insights into the known link between shift or night work and reproductive disorders.

"This research offers some insight into why some women cannot bring pregnancies to full term," said Gerald Weissmann, M.D., Editor-in-Chief of The

FASEB Journal, "and it shows that the womb has a body clock of its own, and that this clock needs to synchronize with the mother's."

The full article is available here: http://www.fasebj.org/content/29/4/1603.full

http://www.eurekalert.org/pub_releases/2015-04/foas-ccd040115.php

Common cholesterol drug stimulates the same receptors as marijuana

New research in The FASEB Journal suggests that fenofibrate activates cannabinoid receptors and may become a viable treatment option for relieving pain, stimulating appetite, reducing nausea and preventing depression

If you want the benefits of medical marijuana without the "unwanted side effects" of cannabis, new research should leave you on a high note. According to a research report appearing in the April 2015 issue of The FASEB Journal, fenofibrate, also known by the brand name Tricor®, may benefit a wide range of health issues, such as pain, appetite stimulation, nausea, as well as immune and various psychiatric and neurological conditions. This suggests that fenofibrate may be the starting point for a new class of cannabis-like drugs to treat these types of conditions.

"By illustrating the relationship between fenofibrate and the cannabinoid system, we aim to improve our understanding of this clinically important drug," said Richard S. Priestley, Ph.D., a researcher involved in the work from the School of Life Sciences at the University of Nottingham Medical School in Nottingham, United Kingdom. "Our study provides the basis for the investigation of new drugs targeting these important receptors."

To make this discovery Priestly and colleagues cultured cells containing cannabinoid receptors and exposed them to a tracer compound, which binds to cannabinoid receptors. They found that fenofibrate was able to displace the tracer, suggesting that it also binds to the receptors. Furthermore, they discovered that fenofibrate actually switched the cannabinoid receptors "on," not only in these cells, but also in sections of intestine. This led to the relaxation of the tissue in a way that mimicked what marijuana does. Despite the fact that fenofibrate has been used for many years, and its mechanism of action was presumed to be through a completely different family of receptors, this suggests that at least some of the effects of fenofibrate may be controlled by cannabinoid receptors. Furthermore, these cannabinoid receptors may be a future target for drugs used to treat pain and a variety of immune and psychiatric diseases.

"It may be difficult to persuade people in Colorado, Washington, and the District of Columbia that there are people who want the beneficial effects of marijuana without actually getting high," said Gerald Weissmann, M.D., Editor-in-Chief of The FASEB Journal, "but there are people who do not want to get stoned just to get the relief that marijuana brings. This new work suggests that possibility."

Richard S. Priestley, Sarah A. Nickolls, Stephen P. H. Alexander, and David A. Kendall. A potential role for cannabinoid receptors in the therapeutic action of fenofibrate. FASEB J. April 2015 29:1446-1455; doi:10.1096/fj.14-263053 ;
http://www.fasebj.org/content/29/4/1446.abstract

http://www.eurekalert.org/pub_releases/2015-04/asu-cre040115.php

Cancer's relentless evolution

All living things - from dandelions to reindeer - evolve over time. Cancer cells are no exception, and are subject to the two overarching mechanisms described by Charles Darwin: chance mutation and natural selection.

In new research, Carlo Maley, PhD., and his colleagues describe compulsive evolution and dramatic genetic diversity in cells belonging to one of the most treatment-resistant and lethal forms of blood cancer: acute myeloid leukemia (AML). The authors suggest the research may point to new paradigms in both the diagnosis and treatment of aggressive cancers, like AML.

Maley is a researcher at Arizona State University's Biodesign Institute and an assistant professor in ASU's School of Life Sciences. His work focuses on applying principles of evolutionary biology and ecology to the study of cancer.

The group's findings appear in this week's issue of the journal Science Translational Medicine.

The cells, they are a changin'

A tumor is a laboratory for evolutionary processes in which nature experiments with an immense repertoire of variants. Mutations that improve a cell's odds of survival are "selected for," while non-adaptive cells are weeded out in the evolutionary lottery.

Genetic diversity therefore provides cancer cells with a library of possibilities, with some mutations conferring heightened resistance to attack by the body's immune system and others helping malignant cells foil treatments like chemotherapy. Generally speaking, the seriousness of a given cancer diagnosis may be linked with genetic diversity in cancerous cells. High diversity means the cancer has many pathways for outsmarting treatment efforts.

The diagnosis of cancer and study of disease progression is often accomplished by examining a tumor sample containing many billions or even trillions of cells. These are subjected to so-called next generation sequencing, a technique that sifts the vast genetic composite, ferreting out sequence variants (or alleles) caused by mutations in genes. The process then evaluates the frequency of these alleles, using the results to chart disease progression and assess the effectiveness of treatment.

According to Maley, such methods may obscure the true degree of genetic diversity, as well as the manner in which mutations arise. "One issue here is that if a mutation occurs in less than 20 percent of the cells, it's hard to detect by modern methods," he says. For example, because individual cells in the tumor probably carry unique mutations, they would be virtually impossible to observe with standard sequencing methods. A further issue is that tracking mutations through bulk analysis of cells is typically based on certain assumptions as to how mutations arise and what their frequencies mean.

A new window

The current study attempts to provide a more accurate picture of what is taking place at the genetic level when an AML patient has a relapse or metastasis of the disease. Rather than carry out conventional bulk analysis of cells, the research group examined individual cells, screening them for the presence of two critical gene mutations common in AML, known as FLT3 and NPM1.

The results significantly alter existing assumptions of cancer progression, indicating much greater genetic diversity in AML than previously assumed. The process of convergent evolution, in which separate lineages develop similar features, appears to account for some of the observed diversity. The researchers found evidence that the exact same mutation was occuring multiple times within the same patient.

Within the paired chromosomes contained in every cell, mutations occurring on one chromosome are known as heterozygous, while those occurring on both are homozygous. The new study shows that in AML, every possible combination of homozygous and heterozygous mutation occurs for the two gene mutations under study.

The study examined individual cells in six patients with AML. The results clearly showed all combinations of homo and heterozygous mutation. "There's no way to explain that with each mutation only happening once," Maley says. Instead, some mutations are occurring repeatedly in the same tumor. "That's scary because it means that these cancers have access to many mutations and can find the same mutation over and over."

Maley notes that influences from the environment may drive convergent evolution but that identical mutations can also arise through pure coincidence, simply by virtue of the enormous numbers involved.

A 1 cm3 AML tumor, for example, may contain a billion cells, each containing some 3 billion base pairs in its genome. Mutations are estimated to occur at a rate of 1 mutation in every billion base pairs. "That means every time the population of cells in a 1 cm tumor undergoes 1 generation, which we think takes just a couple days, every possible mutation of the genome is happening somewhere in that tumor," Maley says. This alone would lead to the same mutation likely occurring independently multiple times.

Curbing cancer's lethality

Given AML's near-limitless capacity for creating novel variants, what can clinicians do to halt the disease's pitiless advance? According to Maley, one hopeful approach would be to use cancer's evolveability to advantage, rather than attempt to fight it head on. "Can we put pressures on the tumor that select for a behavior that we want - a manageable cancer that doesn't kill us?"

This new paradigm draws on a branch of ecology known as life history theory. The idea is to carefully study the environmental factors that may lead organisms to favor either a fast reproducing or slow reproducing strategy to maximize their survivability.

Currently, most cancer therapy relies on frontal assaults on malignant cells. The approach is effective provided the given cancer is limited in the genetic variants it can produce in order to adapt to changing environments and survive. For a cancer with very high genetic diversity (like AML) however, the unintended effect of treatment is often to select for the most aggressive, resistant cells, clearing away their competitors and furnishing them with all the resources they need to flourish.

According to life history theory, fast reproduction tends to occur in environments with high extrinsic mortality. Aggressive cancer treatment creates just such an environment, favoring those cells able to reproduce quickly, producing large numbers of daughter cells, with a few evading extrinsic mortality to repopulate the tumor. On the other hand, a very stable environment often favors slow reproduction, because organisms reach a carrying capacity of their surrounding environment. In this case, the limiting factor becomes competition between like organisms. Here, a slow reproducing strategy favoring greater investment in maintenance and survivability wins the competition.

As Maley points out, the clinical implications are clear: "This approach would say 'let's keep tumors as stable as possible and keep their resources limited.' If we are able to keep the tumor cells contained and let them fight it out, we would expect to see more competitively fit cells that are growing very slowly."

While the current single-cell analysis evaluated just two mutations in AML, the results demonstrated the staggering evoleability of this form of cancer. Eventually, researchers like Maley would like to examine whole genomes in single cells. Presently, many technical hurdles exist. Nevertheless, evolutionary approaches to cancer are already suggesting a broad rethinking of this complex of diseases.

Single-cell genotyping demonstrates complex clonal diversity in acute myeloid leukemia
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http://www.eurekalert.org/pub_releases/2015-04/uoef-eer040215.php

Eating eggs reduces risk of type 2 diabetes

Egg consumption may reduce the risk of type 2 diabetes, according to new research from the University of Eastern Finland.

The findings were published in American Journal of Clinical Nutrition.

Type 2 diabetes is becoming increasingly widespread throughout the world. Research has shown that lifestyle habits, such as exercise and nutrition, play a crucial role in the development of the disease. In some studies, high-cholesterol diets have been associated with disturbances in glucose metabolism and risk of type 2 diabetes. In contrast, in some experimental studies, the consumption of eggs has led to improved glucose balance, among other things. However, there is no experimental data available on the effects of egg consumption on the incidence of type 2 diabetes. In population-based studies, too, the association between egg consumption and type 2 diabetes has been investigated only scarcely, and the findings have been inconclusive. Egg consumption has either been associated with an elevated risk, or no association has been found.

The dietary habits of 2,332 men aged between 42 and 60 years were assessed at the baseline of the Kuopio Ischaemic Heart Disease Risk Factor Study, KIHD, at the University of Eastern Finland in 1984-1989. During a follow-up of 19.3 years, 432 men were diagnosed with type 2 diabetes.

The study found that egg consumption was associated with a lower risk of type 2 diabetes as well as with lower blood glucose levels. Men who ate approximately four eggs per week had a 37 per cent lower risk of type 2 diabetes than men who only ate approximately one egg per week. This association persisted even after possible confounding factors such as physical activity, body mass index, smoking and consumption of fruits and vegetables were taken into consideration. The consumption of more than four eggs did not bring any significant additional benefits.

A possible explanation is that unlike in many other populations, egg consumption in Finland is not strongly associated with unhealthy lifestyle habits such as smoking, low physical activity or consumption of processed meats. In addition to cholesterol, eggs contain many beneficial nutrients that can have an effect on, for example, glucose metabolism and low-grade inflammation, and thus lower the risk of type 2 diabetes. The study also suggests that the overall health effects of foods are difficult to anticipate based on an individual nutrient such as cholesterol alone. Indeed, instead of focusing on individual nutrients, nutrition research has increasingly focused on the health effects of whole foods and diets over the past few years.

Egg consumption and risk of incident type 2 diabetes in men: The Kuopio Ischaemic Heart Disease Risk Factor Study. Jyrki K. Virtanen, Jaakko Mursu, Tomi-Pekka Tuomainen, Heli E.K. Virtanen, Sari Voutilainen . American Journal of Clinical Nutrition. Published online ahead of print, April 2, 2015. Link: http://ajcn.nutrition.org/content/early/2015/04/01/ajcn.114.104109.abstract

http://bit.ly/19OI8Rk

Mothers Who Eat a Newborn's Placenta May or May Not Benefit

Proponents of the practice say it can help relieve postpartum depression, but there are no data to back their assertions

By Rebecca Harrington

New mothers who eat their babies' placentas soon after childbirth are part of a growing fad. Web sites offer recipes and services, such as turning the placenta into a pill, to make the experience more palatable. Proponents claim the practice, known as placentophagy, increases their energy and can even ward off postpartum depression. Although the placenta is packed with nutrients and hormones that help the baby develop and survive in the womb, it can also harbor potentially harmful bacteria and waste products. To date no scientific studies have documented the benefits or risks that may come from eating the placenta.

Scientific American asked Rebecca Baergen about the medical evidence on placentophagy. Baergen is a professor of pathology and laboratory medicine and chief of perinatal and obstetric pathology at New York–Presbyterian Hospital/Weill Cornell Medical Center. She has studied the placenta for more than two decades after training with the pioneer of placental pathology, Kurt Benirschke. ***[An edited transcript of the interview follows.]***

What is the evolutionary basis for placentophagy? Do other animals practice it?

Yes, many mammals that have placentas do this. The mothers do eat the placenta. And that's really part of the justification that people use. They say, "Well, the animals do it, so that's something that we should do." But there are a lot of other things that animals do that we don't do.

It probably has a lot to do with the fact that the animals are out in the wild. If they don't eat the placenta, then scavengers and predators will come around and see or smell the blood. It's kind of an issue of cleanup for those animals, so they don't leave behind a signature for predators that can prey on the young.

Why do some people advocate eating the placenta? What are the demonstrated benefits?

A lot of women have reported a benefit but no studies have been done that really document any kind of beneficial effects. A lot of this could be a placebo effect. We don't know - it's all anecdotal. People give reports saying, "Yes, I've done this

and it had a great benefit. Yes it helped me and it was wonderful." So there are a lot of women who are encouraged to do it.

A lot of what's claimed is that the women feel healthier; they feel stronger. It's very subjective.

It's not something that I personally would recommend. But I'm not necessarily going to say, "No, I don't think you should do that," because I don't have any proof saying it's actually harmful to anyone, either.

What are some of the health risks that could occur?

A lot of placentas have infections, bacterial infections. We had one case here where the mother wanted to take the placenta [but] that request was denied. There was infection in the placenta and in addition there was evidence of meconium - that's fetal feces, basically - which is a waste product that maybe is not necessarily a good thing to be ingesting either. You don't really know in a lot of cases whether that's present.

Probably the main thing is infection. That would be much more of a risk if it's somebody else's placenta, but still remains a risk even if the mother eats her baby's placenta. The placenta is a fetal organ; it belongs to the fetus. It consists of fetal tissue, not maternal tissue. There is maternal blood in it but it's fetal tissue.

Is there any evidence that shows eating the placenta can defend against postpartum depression?

It hasn't been documented. But it's thought that at least some of the postpartum depression has to do with the fact that you have all these hormones produced by the placenta during pregnancy, and then after the baby is delivered that source of hormones is gone. It's thought that that drop in the hormones and maybe other things that are produced by the placenta during pregnancy causes postpartum depression in [mothers] who might have a propensity for that. So if you replace that by taking what was in the placenta, that might alleviate the depression. Again, it has not been proven but it does seem reasonable.

If I was concerned about postpartum depression and I had an issue with that, I would get it treated by modern medicine rather than using a method that's not necessarily proved. I would want to see the scientific evidence before I would consider doing that.

Are mothers allowed to take their placenta from the hospital or are there any regulations preventing that?

It is up to the individual hospital's policy. It's really variable. Some of it has to do with the health statutes. The Joint Commission on accreditation of hospitals says normal placentas from normal deliveries don't have to go to pathology. They do need to be sent when there's a problem with the baby or a problem with the pregnancy or a problem with the delivery because the placenta is actually very important in explaining what happened.

Many hospitals do release placentas to patients at their request, as long as there's not an indication for it to be examined for pathology. It's a complicated issue.

What do you think would be the best ways to ingest it then - encapsulated, raw, incorporated into something?

Some people talk about actually putting the placenta in recipes, cooking and eating it that way. If that's done, you probably are destroying any of the potential benefits that you might be getting, because a lot of the proteins and hormones and blood products are broken down by cooking. It's not like meat. Meat is really muscle, and the placenta is not. From the point of preserving things, if it was not cooked, I think that would probably be better, although it would be safer to have it cooked.

I think when you're dealing with it raw, as long as it's your own placenta, I suppose that would not be [horrible]. You still might be ingesting some potentially unappetizing things. Who would want to ingest feces and infections with inflammatory cells or bacteria? Encapsulation, where they actually take the placental extract and put it into pill form, [has made the practice] popular because it's very palatable without having to actually cook it. I think that makes it a lot easier for people to deal with it. But I personally would not ever want to do it.

What should be done is that the people who strongly believe in it should try to get studies done that will document the effects. If there really is a benefit, then these studies will show that.

http://www.eurekalert.org/pub_releases/2015-04/cp-wgc032615.php

With geomagnetic compass hooked to the brain, blind rats act like they can see

Findings show the incredible flexibility of the mammalian brain

By attaching a microstimulator and geomagnetic compass to the brains of blind rats, researchers reporting in the Cell Press journal Current Biology on April 2 found that the animals can spontaneously learn to use new information about their location to navigate through a maze nearly as well as normally sighted rats. Researchers say the findings suggest that a similar kind of neuroprosthesis might also help blind people walk freely through the world.

Most notably, perhaps, the findings show the incredible flexibility of the mammalian brain.

"The most remarkable point of this paper is to show the potential, or the latent ability, of the brain," says Yuji Ikegaya of the University of Tokyo. "That is, we demonstrated that the mammalian brain is flexible even in adulthood - enough to

adaptively incorporate a novel, never-experienced, non-inherent modality into the pre-existing information sources."

In other words, he says, the brains of the animals they studied were ready and willing to fill in "the 'world' drawn by the five senses" with a new sensory input.

What Ikegaya and his colleague Hiroaki Norimoto set out to do was to restore not vision per se, but the blind rats' allocentric sense. That sense is what allows animals and people to recognize the position of their body within the environment. What would happen, the researchers asked, if the animals could "see" a geomagnetic signal? Could that signal fill in for the animals' lost sight? Would the animals know what to do with the information?

The head-mountable geomagnetic sensor device the researchers devised allowed them to connect a digital compass (the kind you'd find in any smart phone) to two tungsten microelectrodes for stimulating the visual cortex of the brain. The very lightweight device also allowed the researchers to turn the brain stimulation up or down and included a rechargeable battery.

Once attached, the sensor automatically detected the animal's head direction and generated electrical stimulation pulses indicating which direction they were facing - north or south, for instance.

The "blind" rats were then trained to seek food pellets in a T-shaped or a more complicated maze. Within tens of trials, the researchers report, the animals learned to use the geomagnetic information to solve the mazes. In fact, their performance levels and navigation strategies were similar to those of normally sighted rats. The animals' allocentric sense was restored.

"We were surprised that rats can comprehend a new sense that had never been experienced or 'explained by anybody' and can learn to use it in behavioral tasks within only two to three days," Ikegaya says.

The findings suggest one very simple application: to attach geomagnetic sensors to the canes used by some blind people to get around. More broadly, the researchers expect, based on the findings, that humans could expand their senses through artificial sensors that detect geomagnetic input, ultraviolet radiation, ultrasound waves, and more.

Our brains, it appears, are capable of much more than our limited senses allow.

"Perhaps you do not yet make full use of your brain," Ikegaya says. "The limitation does not come from your lack of effort, but it does come from the poor sensory organs of your body. The real sensory world must be much more 'colorful' than what you are currently experiencing."

Current Biology, Norimoto et al.: "Visual Cortical Prosthesis with a Geomagnetic Compass Restores Spatial Navigation in Blind Rats"

http://bit.ly/1IdtDpf

Artificial Sweeteners May Change Our Gut Bacteria in Dangerous Ways

Substances such as saccharin may alter the type of bacteria inside us, could lead to obesity

Mar 17, 2015 |By Ellen Ruppel Shell

Many of us, particularly those who prefer to eat our cake and look like we have not done so, have a love-hate relationship with artificial sweeteners. These seemingly magical molecules deliver a dulcet taste without its customary caloric punch. We guzzle enormous quantities of these chemicals, mostly in the form of aspartame, sucralose and saccharin, which are used to enliven the flavor of everything from Diet Coke to toothpaste. Yet there are worries. Many suspect that all this sweetness comes at some hidden cost to our health, although science has only pointed at vague links to problems.

Last year, though, a team of Israeli scientists put together a stronger case. The researchers concluded from studies of mice that ingesting artificial sweeteners might lead to - of all things - obesity and related ailments such as diabetes. This study was not the first to note this link in animals, but it was the first to find evidence of a plausible cause: the sweeteners appear to change the population of intestinal bacteria that direct metabolism, the conversion of food to energy or stored fuel. And this result suggests the connection might also exist in humans.

In humans, as well as mice, the ability to digest and extract energy from our food is determined not only by our genes but also by the activity of the trillions of microbes that dwell within our digestive tract; collectively, these bacteria are known as the gut microbiome. The Israeli study suggests that artificial sweeteners enhance the populations of gut bacteria that are more efficient at pulling energy from our food and turning that energy into fat. In other words, artificial sweeteners may favor the growth of bacteria that make more calories available to us, calories that can then find their way to our hips, thighs and midriffs, says Peter Turnbaugh of the University of California, San Francisco, an expert on the interplay of bacteria and metabolism.

Bacterial gluttons

In the Israeli experiment, 10-week-old mice were fed a daily dose of aspartame, sucralose or saccharin. Another cluster of mice were given water laced with one of two natural sugars, glucose or sucrose. After 11 weeks, the mice receiving sugar were doing fine, whereas the mice fed artificial sweeteners had abnormally high blood sugar (glucose) levels, an indication that their tissues were having difficulty absorbing glucose from the blood. Left unchecked, this “glucose intolerance” can lead to a host of health problems, including diabetes and a

heightened risk of liver and heart disease. But it is reversible: after the mice were treated with broad-spectrum antibiotics to kill all their gut bacteria, the microbial population eventually returned to its original makeup and balance, as did blood glucose control.

“These bacteria are not agnostic to artificial sweeteners,” says computational biologist Eran Segal of the Weizmann Institute of Science in Rehovot, Israel, one of the two scientists leading the study. The investigators also found that the microbial populations that thrived on artificial sweeteners were the very same ones shown - by other researchers - to be particularly abundant in the guts of genetically obese mice.

Jeffrey Gordon, a physician and biologist at Washington University in St. Louis, has done research showing that this relation between bacteria and obesity is more than a coincidence. Gordon notes that more than 90 percent of the bacterial species in the gut come from just two subgroups - Bacteroidetes and Firmicutes. Gordon and his team found several years ago that genetically obese mice (the animals lacked the ability to make leptin, a hormone that limits appetite) had 50 percent fewer Bacteroidetes bacteria and 50 percent more Firmicutes bacteria than normal mice did. When they transferred a sample of the Firmicutes bacterial population from the obese mice into normal-weight ones, the normal mice became fatter. The reason for this response, Gordon says, was twofold: Firmicutes bacteria transplanted from the fat mice produced more of the enzymes that helped the animals extract more energy from their food, and the bacteria also manipulated the genes of the normal mice in ways that triggered the storage of fat rather than its breakdown for energy.

Gordon believes something similar occurs in obese humans. He found that the proportion of Bacteroidetes to Firmicutes bacteria increases as fat people lose weight through either a low-fat or low-carbohydrate diet. Stanford University microbiologist David Relman says this finding suggests that the bacteria in the human gut may not only influence our ability to extract calories and store energy from our diet but also have an impact on the balance of hormones, such as leptin, that shape our very eating behavior, leading some of us to eat more than others in any given situation.

The burning question, of course, is whether artificial sweeteners can truly make humans sick and fat. Segal thinks they probably do, at least in some cases. He and his team analyzed a database of 381 men and women and found that those who used artificial sweeteners were more likely than others to be overweight. They were also more likely to have impaired glucose tolerance. Obesity is, in fact, well known as a risk factor for the development of glucose intolerance as well as more severe glucose-related ailments, such as diabetes.

These patterns do not prove that the sweeteners caused the problems. Indeed, it is quite possible that overweight people are simply more likely than others to consume artificial sweeteners. But Segal's team went further, testing the association directly in a small group of lean and healthy human volunteers who normally eschewed artificial sweeteners. After consuming the U.S. Food and Drug Administration's maximum dose of saccharin over a period of five days, four of the seven subjects showed a reduced glucose response in addition to an abrupt change in their gut microbes. The three volunteers whose glucose tolerance did not dip showed no change in their gut microbes.

Although not everyone seems susceptible to this effect, the findings do warrant more research, the scientists say. The Israeli group concluded in its paper that artificial sweeteners “may have directly contributed to enhancing the exact epidemic that they themselves were intended to fight” - that is, the sweeteners may be making at least some of us heavier and more ill.

A cause-and-effect chain from sweeteners to microbes to obesity could explain some puzzles about obese people, says New York University gastroenterologist Ilseung Cho, who researches the role of gut bacteria in human disorders. He points out that in studies, most people who switch from sugar to low-calorie sweeteners in an effort to lose weight fail to do so at the expected rate. “We've suspected for years that changes in gut bacteria may play some role in obesity,” he says, although it has been hard to pinpoint this effect. But Cho adds that it is clear that “whatever your normal diet is can have a huge impact on the bacterial population of your gut, an impact that is hard to overestimate. We know that we don't see the weight-loss benefit one would expect from these nonnutritive sweeteners, and a shift in the balance of gut bacteria may well be the reason, especially a shift that results in a change in hormonal balances. A hormone is like a force multiplier - and if a change in our gut microbes has an impact on hormones that control eating, well, that would explain a lot.”

Microbes vs. genes

Naturally there are many questions left to answer. Cathryn Nagler, a pathologist at the University of Chicago and an expert on gut bacteria and food allergies, says that the enormous genetic variations in humans make extrapolations from mice suspect. “Still, I found the data very compelling,” she says of the Israeli artificial sweetener study. Relman agrees that rodent studies are not always reflective of what happens in humans. “Animal studies can point to a general phenomenon, but animals in these studies tend to be genetically identical, while in humans, lifestyle histories and genetic differences can play a very powerful role,” he says. The constellation of microbes in a human body is a reflection of that body's particular history - both genetic and environmental.

"The microbiome is a component intertwined in a complex puzzle," Relman continues. "And sometimes the genetics is so strong that it will override and drive back the microbiota." Genetic variations might explain why only four of the seven saccharin-fed humans had a change in their gut bacteria, for instance, although genetics is only one of a number of possible factors. And if someone is genetically predisposed to obesity and consumes a diet that promotes that obesity, the microbes might change to take advantage of that diet, thereby amplifying the effect.

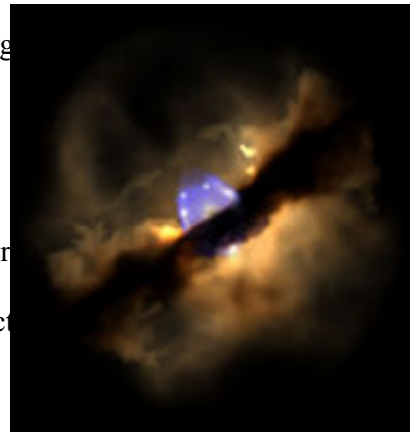
The Israeli researchers agree that it is far too soon to conclude that artificial sweeteners cause metabolic disorders, but they and other scientists are convinced that at least one - saccharin - has a significant effect on the balance of microbes in the human gut. "The evidence is very compelling," Turnbaugh says. "Something is definitely going on." Segal, for one, is taking no chances: he says that he has switched from using artificial to natural sweetener in his morning coffee.

http://www.bbc.com/news/science-environment-32168507

Star's birth glimpsed 'in real time'

Astronomers have witnessed a key stage in the birth of a very heavy star, using two radio telescope views of the process taken 18 years apart.

The young star is 4,200 light-years from Earth and appears to be surrounded by a doughnut-shaped cloud of dust. That cloud slows down the hot, ionised wind that the star blasts into space, causing it to form an elongated column perpendicular to the dusty ring.



The new results represent "before and after" glimpses of that column forming. They were captured by the Very Large Array, a battery of 27 antennae in the New Mexico desert, and are published in the journal Science.

"The comparison is remarkable," said first author Carlos Carrasco-Gonzalez, from the National Autonomous University of Mexico. The compact rounded wind indicated by data from 1996 transforms - just 18 years later in 2014 - into a "distinctly elongated outflow".

Data from 1996 - illustrated here as a 3D simulation - showed a compact, round blast of wind (in blue)

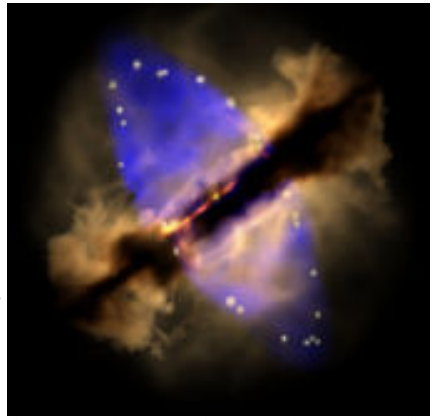
One to watch

The infant star is about 300 times brighter than the Sun and goes by the catchy name of W75N(B)-VLA2. Being able to observe its dramatic growing pains in real time is unique, according to Prof Huib van Langevelde from Leiden University in the Netherlands, another of the study's authors.

"This object is providing us an exciting opportunity to watch the developments over the next few years, as this very young star develops the characteristic bipolar outflow morphology," said Prof van Langevelde, who also works at the Joint Institute for VLBI in Europe (JIVE).

VLBI - very long baseline interferometry - is the method of comparing signals between widely-spaced antennae, effectively simulating one massive telescope.

One of the major findings that has already emerged from studying W75N(B)-VLA2 relates to earlier work led by JIVE scientists, who in 2009 traced the large-scale magnetic field in that region of space and reported that the field surrounding the young star was neatly aligned with it.



Now, it seems the elongated outflow that has burst forth in just 18 years is also aligned with that magnetic field - suggesting that magnetism is playing a crucial role in the star's formation. The team hopes to watch and learn more as the "protostar" continues its turbulent development.

The 2014 data revealed the wind to be much more elongated, emerging from the presumed ring of dust

"Our understanding of how massive young stars develop is much less complete than our understanding of how Sun-like stars develop," said Dr Gabriele Surcis, another co-author from JIVE. "It's going to be really great to be able to watch one as it changes."

http://bit.ly/1P7yYjI

Test Tube Burgers Get a $324,989 Price Cut

The scientists behind lab-grown meat think they can soon offer it at a price most of us can actually afford

By Samantha Larson

When scientists unveiled the first lab-grown burger in 2013, they had a strong argument that it represented the future of meat. Test-tube protein could revolutionize our current agricultural system - and address some of environmental and ethical problems with the world's massive amount of meat consumption. But critics also had a very strong argument as to why this might not be a panacea: that single patty cost a whopping $325,000 to produce.

But "schmeat" scientists have not given up on bringing their goods to the masses. In fact, professor Mark Post told ABC Australia he estimates he will soon be able to cut costs down to just about $11 per burger.

Why was the piece of beef so expensive to start with? For one, it took a lot of the scientists' time to figure out how to make. Vice Munchies explains the complicated process behind how it was eventually done:

The 2013 burger was made by taking muscle cells from a cow, using it to cultivate stem cells, and then fusing them with collagen. Then, electricity was used to stimulate the subsequent 'muscle' strands, causing them to flex in a way that would render them meatier and more similar to conventional beef. Sounds like a breeze, right? Then consider that 20,000 of these individual strands would need to be cultivated, processed, and seasoned in order to create a single burger.

The tube-meat enthusiasts are not dissuaded by the high price tag. "Consider it like the very first computer," Isha Datar, director of a nonprofit dedicated to the development of lab-grown meat alternatives, told Grist. "It was very exclusive and impractical in every sense – it was not something you'll ever see in a store." But now - to continue the computer metaphor - computers of all shapes and sizes are completely run-of-the-mill. We even have computers that fit in our pockets.

"I do think that in 20, 30 years from now we will have a viable industry producing alternative beef," Post said.

Cost aside, there is another factor to consider: whether schmeat actually tastes good. Because only three people got to sample the first one, most of us can't know for sure - at least not for awhile. But, going off the taste-testers comments, it sounds like there is still some work to be done. "The testers reported that the burger tasted almost like a real one, but not as juicy and 'surprisingly crunchy,'" reports MIT Technology Review. And "crunchy" is...not exactly a desirable quality in meat.

As Datar explained to Grist, flavor comes down to being able to culture other cell types, like fat and blood - not just muscle. Hopefully, figuring out how to produce those cells won't cost too much.

http://www.medscape.com/viewarticle/842260

A New Era in Pharmacotherapy

Recent approval of the first biosimilar agent in the United States

Jonathan Kay, MD

Hello. I'm Dr Jonathan Kay, professor of medicine at the University of Massachusetts Medical School and director of clinical research in the Division of Rheumatology at the University of Massachusetts Memorial Medical Center, both in Worcester, Massachusetts.

I would like to talk about the recent approval of the first biosimilar agent in the United States. On March 6, 2015, the US Food and Drug Administration (FDA) approved Zarxio™ (filgrastim-sndz),[1] a biosimilar filgrastim, for use in treating neutropenia in a number of situations including for patients receiving chemotherapy; patients having hematologic cell mobilization; and patients with absolute neutropenia. This biosimilar filgrastim is essentially identical in amino acid structure and highly similar in other aspects of its chemical structure to Neupogen® (filgrastim), the agent that has been produced and marketed by Amgen since 1991.[1]

The FDA approved this biosimilar filgrastim on the basis of a totality of evidence including analytical studies that demonstrated identical amino acid sequence to Neupogen; highly similar chemical structures; animal toxicology - which demonstrated highly similar toxicologic profiles; pharmacokinetic and pharmacodynamic studies in humans using an absolute neutrophil count as the pharmacodynamic marker; and a single clinical trial conducted with 218 patients with breast cancer receiving chemotherapy where Zarxio and Neupogen had equivalent number of days with absolute neutropenia and similar safety profiles.[1,2] The approval of this first biosimilar in the United States is likely to be followed shortly by the approval, or at least the review, of other biosimilar molecules.[3]

The monoclonal antibody CT-P13, a biosimilar infliximab, was to have been reviewed by the FDA on March 17, 2015 - but this hearing has been postponed with a request for additional data.[4] The biosimilar infliximab CT-P13 has been approved by the European Medicines Agency[5] for use in the European Union,[6] Japan and Turkey,[7] South Korea,[8] and other countries. It is marketed both as Remsima™ and Inflectra™ and has been available since 2013 in the European Union.[6]

I just returned from a visit to Slovakia, where I spoke with rheumatologists who have been using the biosimilar monoclonal antibody CT-P13. There it is marketed both as Remsima by Celltrion and Inflectra by Hospira.[9] They said that this monoclonal antibody is effective and has demonstrated no clinically significant difference from Remicade®, the originator biopharmaceutical against which CT-P13 was compared in clinical trials.[5]

This is the beginning of a new era, an exciting time in the United States where biosimilars are *here now*. I look forward to watching the review and approval of additional biosimilars and look forward to seeing you again on Medscape. Thank you. Please leave your comments below.

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New evidence shows carbon's importance to ocean life's survival 252 million years ago

First demonstration of how elemental carbon became an important construction material of some forms of ocean life

ARLINGTON, Texas - A new study led by scientists with The University of Texas at Arlington demonstrates for the first time how elemental carbon became an important construction material of some forms of ocean life after one of the greatest mass extinctions in the history of Earth more than 252 million years ago.

As the Permian Period of the Paleozoic Era ended and the Triassic Period of the Mesozoic Era began, more than 90 percent of terrestrial and marine species became extinct. Various proposals have been suggested for this extinction event, including extensive volcanic activity, global heating, or even one or more extraterrestrial impacts.

The work is explained in the paper, "High influx of carbon in walls of agglutinated foraminifers during the Permian-Triassic transition in global oceans," which is published in the March edition of International Geology Review.

Researchers focused on a section of the latest Permian aged rocks in Vietnam, just south of the Chinese border, where closely spaced samples were collected and studied from about a four-meter interval in the boundary strata.

Merlynd Nestell, professor of earth and environmental sciences in the UT Arlington College of Science and a co-author of the paper, said there was extensive volcanic activity in both the Northern and the Southern Hemispheres during the Permian-Triassic transition.

"Much of the volcanic activity was connected with the extensive Siberian flood basalt known as the Siberian Traps that emerged through Permian aged coal deposits and, of course, the burning of coal created CO2," Nestell said.

He noted that there was also synchronous volcanic activity in what is now Australia and southern China that could have burned Permian vegetation. The carbon from ash accumulated in the atmosphere and marine environment and was used by some marine microorganisms in the construction of their shells, something they had not done before.

This new discovery documents elemental carbon as being a major construction component of the tiny shells of single-celled agglutinated foraminifers, ostracodes, and worm tubes that made up part of the very limited population of bottom-dwelling marine organisms surviving the extinction event.

"Specimens of the boundary interval foraminifers seen in slices of rock that were ground thin and studied from other places in the world revealed black layers," said Galina P. Nestell, study co-author and adjunct research professor of earth and environmental sciences at UT Arlington. "But nobody really checked the composition of the black material."

Nestell said this phenomenon has never been reported although sequences of rocks that cross this important Permian-Triassic boundary have been studied in Iran, Hungary, China, Turkey, Slovenia and many other parts of the world.

For the study, Asish Basu, chair of earth and environmental sciences at UT Arlington, analyzed clusters of iron pyrite attached to the walls of the foraminifer shells for lead isotopes. Data from these pyrite clusters support the presence of products of coal combustion that contributed to the high input of carbon into the marine environment immediately after the extinction event.

Brooks Ellwood, emeritus professor of Earth and Environmental Sciences at UT Arlington and a professor in the Louisiana State University Department of

Geology and Geophysics, collected the samples to study the Permian-Triassic boundary interval using magnetic and geochemical properties. He and his colleague Luu Thi Phuong Lan of the Vietnamese Academy of Science and Technology in Hanoi, Vietnam, also collected the samples used in the biostratigraphic work by the Nestells and Bruce Wardlaw of the Eastern Geology and Paleoclimate Science Center at the U.S. Geological Survey and adjunct professor at UT Arlington.

By using time-series analysis of magnetic measurements, Ellwood discovered the extinction event to have lasted about 28,000 years. It ended about 91,000 years before the actual Permian-Triassic boundary level - as defined worldwide by the first appearance of the fossil conodont species Hindeodus parvus - identification done by Wardlaw.

Galina Nestell said the high carbon levels began after the extinction event about 82,000 years before the official boundary horizon and continued until about 3,000 years after the Permian-Triassic boundary horizon. The boundary horizon is calculated to be 252.2 million years before present.

Other co-authors who contributed to parts of the study include Andrew Hunt, EES associate professor at UT Arlington, Nilotpal Ghosh of the University of Rochester; Harry Rowe of the Bureau of Economic Geology at the University of Texas at Austin; Jonathan Tomkin of the University of Illinois, Urbana; and Kenneth Ratcliffe of Chemostrat Inc. in Houston.

http://bit.ly/1Iy1fuM

Most Plastic Trash Comes From Farms

Here's what they're trying to do about it

By Marissa Fessenden

There once was a great future in plastics, but their waste is weighing that future down. Scraps and bit cumulatively reaching more than 250,000 tons end up in the ocean, and even the smallest particles cause trouble as they clog corals. Eventually some of the six billion tons of plastic manufactured since the mid-20th century becomes a sort of stone, an aggregate of bound plastic and rocks.

Scientists still aren't sure how so much plastic ends up in the ocean, but they do know where much of it starts. Elizabeth Grossman visited a small farm owned by Kara Gilbert to track down agricultural plastics. She reports for Ensia:

On a visit to the four-acre farm on lush Sauvie Island at the confluence of the Willamette and Columbia Rivers near Portland, Ore., Gilbert gives me a tour de farm plastics. The fields are just being readied for the season, but black plastic is already laid out under a hoop house. PVC water pipes are being set into place and drip irrigation tape is ready to be deployed, as are plastic sacks of fertilizer. Out in the greening field, little orange-pink plastic plant tags on ankle-high stakes flap in the wet breeze to mark rows of just-sprouted peas.

This tiny produce farm buys between $4,000 and $6,000 worth of plastic every year, Grossman writes. Multiply that number times the number of farms and keep in mind that the larger farms will use much more… you get the picture. Bales are wrapped, greenhouses covered, pesticides stored all in plastic. Gene Jones of the Southern Waste Information eXchange estimates that the U.S. uses about one billion pounds of plastic in agriculture every year.

Fortunately, we are trying to do better. Farm plastics are no longer burned or buried on farm property, or at least most states ban the practice. Now growers are trying to use less plastic by reusing when they can. Grossman writes:

By far the biggest opportunity to reduce farm plastic waste, however, is through recycling. Currently only about 10 percent of farm plastics are recycled. Increasing that number will depend on making drop-off more convenient and expanding options for giving plastic a second life.

In New York, where a statewide ban on backyard or farm burning of plastics was passed in 2009, the Cornell program worked with the state's Department of Environmental Conservation to pioneer agricultural plastics recycling and do educational outreach about recycling options through extension programs and local soil and water conservation districts.

But recycling farm plastic can be challenging. Grossman spoke to an Oregon-based company that recycles baling twine, the orange plastic rope that keeps hay bales together. Apparently the material is so abrasive that many machines can't handle it. Workers have to remove the pieces of hay still clinging to the twine painstakingly by hand. Another company makes reusable grocery bags from ag plastics. A third processes old irrigation pipes into pellets that can be used to make plastic sheets and films for growing produce.

The use of biodegradable plastics might also help - a Washington State University publication cites the benefits of plant starch-based mulches, as opposed to petroleum-based plastic mulches, used for weed suppression and to keep soil warm and moist for growing crops.

Like many multifaceted issues, the problems posed by ag plastics won't have one solution. Hopefully we can have many creative solutions, such as the Netherlands' plan to nab plastic before it escapes to sea and build floating parks for humans above and fish and sea creatures below.

http://bit.ly/1NIWQXv

Here's How Europeans Quickly Evolved Lighter Skin

Darker skinned people lived in Europe until fairly recently

By Marissa Fessenden

As Europeans divided and conquered much of the world, they carried the genes for light skin with them. But even Europeans haven't been white for very long.

New analysis of ancient European genes shows that other traits we associate with modern Europeans, such as tallness and the ability to digest milk, are also relatively recent additions to the continent's genetic profile.

The new data, presented at the annual meeting of the American Association of Physical Anthropologists, comes from the genomes of 83 people found in archeological sites across Europe, reports Ann Gibbbons for Science.

For years, researchers assumed that skin lightened as humans migrated from Africa and the Middle East into Europe, about 40,000 years ago.

A sun lower in the sky and shorter day lengths would have favored skin that more easily synthesized vitamin D. But researchers are now learning that other factors must have been at play.

For example, earlier this year, the genome sequencing of a hunter-gatherer who lived in what is now Spain helped build the case that Europe was home to blue-eyed but dark-skinned people.

This man, however, lived just 7,000 years ago. The researchers write that their analysis suggests that light skin was not yet widespread and ubiquitous in Europe at the time.

Earlier work done with the genes of the 83 people in the new study, supported by linguistic evidence, also shows that populations in Europe about 8,000 years ago would have been mixed and diverse.

The new study adds to this growing pile of evidence. Gibbons reports that the researchers found that Europeans probably couldn't have digested milk until about 4,300 years ago. And the story of skin pigmentation is complex. She writes:

[T]he new data confirm that about 8500 years ago, early hunter-gatherers in Spain, Luxembourg, and Hungary also had darker skin: They lacked versions of two genes - SLC24A5 and SLC45A2 - that lead to depigmentation and, therefore, pale skin in Europeans today.

But in the far north—where low light levels would favor pale skin—the team found a different picture in hunter-gatherers: Seven people from the 7700-year-old Motala archaeological site in southern Sweden had both light skin gene variants, SLC24A5 and SLC45A2. They also had a third gene, HERC2/OCA2, which causes blue eyes and may also contribute to light skin and blond hair. Thus ancient hunter-gatherers of the far north were already pale and blue-eyed, but those of central and southern Europe had darker skin.

"What we thought was a fairly simple picture of the emergence of depigmented skin in Europe is an exciting patchwork of selection as populations disperse into northern latitudes," paleoanthropologist Nina Jablonski, of Penn State told Science.

"This data is fun because it shows how much recent evolution has taken place."

http://bit.ly/1GtmoqW

Nissan aims to put self-driving cars on Japan's road in 2016

Nissan aims to put self-driving cars on Japan's road in 2016

TOKYO - The boss of Nissan wants to put self-driving cars on Japan's roads next year, and says they will be able to navigate busy urban environments on their own by 2020.

Carlos Ghosn, chief executive, said formidable technological and legal challenges remain but that the direction of travel was plain.

"There will be a Nissan product in Japan, which will carry autonomous drive," he told reporters on Thursday at the New York International Auto Show.

"Obviously when you have this kind of technology, you want also the Japanese market to enjoy it as soon as possible."

A five-year tie up with NASA on the technology would see the initial roll out by December 2016, with cars that can drive on highways without anyone at the wheel.

In 2018, models should have the ability to avoid hazards and to change lanes, and by 2020, vehicles should be able to autonomously maneuver through crowded city roads.

"It's going to happen step by step, because we need to make sure that the regulators in the different countries feel comfortable," Ghosn said, according to Kyodo News.

"To persuade the regulators that you can take your hands off the wheel or your eyes from the road is going to take a lot of demonstration."

Nissan, Japan's second biggest automaker, is also looking at working with domestic rivals Toyota and Honda on the technology.

Reports in February said the three are planning to team up with electronics giants and the government in a bid to propel the country into the front ranks of self-driving cars.

The move is part of a government initiative to support domestic industries as competition in the field intensifies globally, with Google testing its own car and Apple also reported to be working on such a vehicle.

The Japanese government has set up a panel to look at the legal issues surrounding autonomous cars, which under current laws are not allowed on public roads.

One of the key factors is that of who bears responsibility in the event of an accident when a car is driving itself.

Advocates of self-driving cars say they could help reduce the number of crashes on the roads because they remove the potential for human error.

More than 4,000 people die in traffic accidents in Japan every year.

http://bit.ly/1yKZ0z4

Next-generation GMOs: Pink pineapples and purple tomatoes

British company planning to apply for U.S. permission to produce and sell purple tomatoes that have high levels of anthocyanins

By MARY CLARE JALONICK

WASHINGTON - Cancer-fighting pink pineapples, heart-healthy purple tomatoes and less fatty vegetable oils may someday be on grocery shelves alongside more traditional products. These genetically engineered foods could receive government approval in the coming years, following the OK given recently given to apples that don't brown and potatoes that don't bruise.

The companies and scientists that have created these foods are hoping that customers will be attracted to the health benefits and convenience and overlook any concerns about genetic engineering. "I think once people see more of the benefits they will become more accepting of the technology," says Michael Firko, who oversees the Agriculture Department's regulation of genetically modified organisms, or GMOs.

Critics aren't so sure. They say there should be more thorough regulation of modified foods, which are grown from seeds engineered in labs, and have called for mandatory labeling of those foods. The Agriculture Department only has the authority to oversee plant health of GMOs, and seeking Food and Drug Administration's safety approval is generally voluntary. "Many of these things can be done through traditional breeding," says Doug Gurian-Sherman of the advocacy group Center for Food Safety. "There needs to be skepticism."

What could be coming next? Del Monte has engineered a pink pineapple that includes lycopene, an antioxidant compound that gives tomatoes their red color and may have a role in preventing cancer. USDA has approved importation of the pineapple, which would be grown only outside of the United States; it is pending FDA approval. A small British company is planning to apply for U.S. permission to produce and sell purple tomatoes that have high levels of anthocyanins, compounds found in blueberries that some studies show lower the risk of cardiovascular disease and cancer. FDA would have to approve any health claims used to sell the products.

Seed giants Monsanto and Dow AgroSciences are separately developing modified soybean, canola and sunflower oils with fewer saturated fats and more Omega-3 fatty acids. The Florida citrus company Southern Gardens is using a spinach gene to develop genetically engineered orange trees that could potentially resist citrus greening disease, which is devastating the Florida orange crop. Okanagan Specialty Fruits Inc., the company that created the non-browning apples, is also looking at genetically engineering peaches, cherries and apples to resist disease and improve quality.

A few genetically engineered fruits and vegetables are already available in grocery stores: Hawaiian papaya, some zucchini and squash, and a small amount of the sweet corn we eat, for example. But the bulk of the nation's genetically engineered crops are corn and soybeans that are eaten by livestock or made into popular processed food ingredients like corn starch, soybean oil or high fructose corn syrup.

The engineered corn and soybeans have faced resistance from environmental groups and some consumers who are wary of the technology, saying not enough is known about it. While science has so far shown that genetically engineered foods are safe, the groups have called for the labeling so consumers know what they are eating. According to a December Associated Press-GfK poll, two-thirds of Americans favor those labels.

Facing that concern, companies developing the new products say their strategy for winning over consumers is to harness the increased interest in healthy eating.

"This is a new wave of crops that have both grower benefits and consumer benefits," says Doug Cole of J.R. Simplot, the company that developed the potatoes. Simplot's potatoes are engineered to have fewer black spots, a benefit not only for farmers seeking higher yields but also for consumers who wouldn't have to soak them before preparation.

British scientist Cathie Martin has developed the modified purple tomatoes and hopes to eventually sell them as a juice in the United States. She says some of those same health-conscious consumers that have concerns over GMOs should be attracted to a product with potential to help lower the risk of cancer.

"This product has been designed to be good for them," Martin says.

Retailers are still uncertain. McDonald's buys Simplot's conventional potato products, but said the company does not have "current plans" to source any GMO potatoes. Other retail chains have already pledged not to sell a genetically engineered salmon that is pending approval at the Food and Drug Administration.

http://www.bbc.com/news/health-32150519

Skin cancer 'linked to package holiday boom'

A boom in cheap package holidays in the 1960s is partly behind the "worrying rise" in skin cancers in pensioners, Cancer Research UK suggests.

By Smitha Mundasad Health reporter, BBC News

The charity says that although all ages are at risk, many older people would not have been aware of how to protect themselves four decades ago.

Figures show that 5,700 over-65s are diagnosed with the condition each year, compared to just 600 in the mid-1970s.

The condition can often be prevented by covering up and avoiding sunburn.

Around 13,300 people are diagnosed with malignant melanoma - the most serious form of skin cancer - each year in the UK. And 2,100 lives are lost to the disease annually.

Numbers are increasing across all age groups but the steepest rise is seen in over-65s.

The charity said all ages are benefitting from public health messages explaining the dangers of holiday sun.

Sue Deans, a 69-year old mother of three, was first diagnosed with skin cancer in 2000 and again in 2007.

She said: "I was part of the generation when package holidays became affordable and you could go abroad nearly every year.

"I don't think there was much understanding at the time about the impact that too much sun can have on your risk of getting skin cancer.

"And I loved the sun but suffered quite a bit of sunburn over the years."

She spotted signs of her cancer early on and has had successful surgery, but remains vigilant for anything that might need further checks.

Professor Richard Marais of Cancer Research UK (CRUK), said: "It is worrying to see melanoma rates increasing at such a fast pace, and across all age groups.

"It is important people keep an eye on their skin and seek medical opinion if they see any changes to their moles or even to normal areas of skin.

"Melanoma is often detected on men's backs and women's legs but can appear on any part of the body."

Research suggests that getting sunburnt just once every two years can increase the odds of developing malignant melanoma.

Dr Julie Sharp, head of health information at CRUK, said: "You can burn at home just as easily as you can on holiday, so remember to spend time in the shade, wear a T-shirt and a hat to protect your skin and regularly apply sunscreen that is at least factor 15 and has four stars."

Johnathon Major, from the British Association of Dermatologists, said: "The increasing incidence of skin cancer within the UK is alarming.

"As people are living longer, more people are reaching an age where they are at a higher risk.

"Interest in package holidays and in fashion tanning are among the reasons that more people are developing skin cancer.

"But it's crucial to remember that you don't have to go on holiday or use a sun bed to heighten your risk. Skin cancers can develop as a result of both short-term and long-term overexposure to the sun's rays within the UK."

http://www.bbc.com/news/uk-32193606

Antibiotic resistance: 80,000 'might die' in future outbreak

The rise of antimicrobial resistance could make currently routine medical procedures "high-risk"

About 80,000 people could die if there were a "widespread outbreak" of an antibiotic-resistant blood infection, according to a government document.

The National Risk Register of Civil Emergencies says such an outbreak could be expected to hit 200,000 people - and two in five of them "might die".

The document also says "high numbers of deaths could also be expected" from other forms of resistant infection.

It warns infection risk could make "much of modern medicine" unsafe.

The Cabinet Office document says the number of infections "complicated" by antimicrobial resistance is expected to "increase markedly over the next 20 years".

"Without effective antibiotics, even minor surgery and routine operations could become high-risk procedures, leading to increased duration of illness and ultimately premature mortality," it says.

It says procedures such as organ transplantation, bowel surgery and some cancer treatments would become unsafe.

'Dark ages'

The document, published last month, adds: "If a widespread outbreak were to occur, we could expect around 200,000 people to be affected by a bacterial blood infection that could not be treated effectively with existing drugs, and around 80,000 of these people might die."

It says the UK government is "leading work with international partners" to tackle this "global problem".

Prime Minister David Cameron has previously warned that the world could be "cast back into the dark ages of medicine" unless action is taken to tackle the threat of resistance to antibiotics.

England's chief medical officer, Dame Sally Davies, has called the problem a "ticking time bomb".

Antibiotic use in the UK has been rising and the National Institute for Health and Care Excellence recently called for doctors to "question" the work of colleagues who prescribe too many.

The Cabinet Office document also rates other threats to the UK both in terms of their anticipated likelihood and their "relative impact" - with a flu pandemic and "catastrophic terrorist attacks" given the highest impact ratings.