http://www.eurekalert.org/pub_releases/2016-06/uoc--avb061616.php

Aspirin versus blood thinners in atrial fibrillation patients with stroke risk

Nearly 40 percent of patients treated with aspirin alone despite previous data showing blood thinners more beneficial

Researchers at University of California San Diego School of Medicine and University of California, San Francisco School of Medicine report that more than 1 in 3 atrial fibrillation (AF) patients at intermediate to high risk for stroke are treated with aspirin alone, despite previous data showing this therapy to be inferior to blood thinners. The findings publish online June 20 in the Journal of the American College of Cardiology.

The study, which examined more than 200,000 AF patients at risk for stroke, found approximately 40 percent were treated with aspirin alone even though previous studies have demonstrated this treatment option is not as beneficial as oral blood thinners, such as warfarin, for reduction of thromboembolism, an obstruction of a blood vessel by a clot that has become dislodged.

The incidence of stroke for AF patients is up to seven times greater than in those without the condition. In AF, electrical impulses in the upper chambers of the heart are chaotic and the atrial walls quiver rather than contract normally in moving blood to the lower chambers. As a result, blood clots may form.

"Stroke prevention is critical to the management of AF patients. However, giving aspirin alone to this population may not be the best treatment therapy because it is either minimally effective or not effective at all and still comes with risks, such as intracranial hemorrhage," said lead author Jonathan C. Hsu, MD, cardiologist at UC San Diego Health and assistant clinical professor of medicine at UC San Diego School of Medicine. "Our study results show a gap in the appropriate treatment of AF patients at risk for stroke. The findings also highlight the critical need for cardiology specialists to adhere to standardized recommendations regarding the use of oral blood thinners instead of aspirin."

Health issues related to coronary artery disease (blockages of the heart arteries), including hypertension, dyslipidemia (abnormal amounts of fats in the blood) or a prior heart attack were associated with more frequent prescription of aspirin only; being male, a higher BMI, a prior stroke and congestive heart failure were associated with more frequent prescription of blood thinners.

"The high rate of an aspirin-only prescription for AF patients with coronary artery disease and other stroke risk factors is concerning," said Hsu. "It appears patients with more risk factors for having a stroke with AF are less likely to get the proper treatment, which is oral blood thinners."

Hsu and his team also found approximately one-third of AF patients in the study without significant coronary artery disease were prescribed both a blood thinner and aspirin, placing them at higher risk for bleeding without any evidence of benefit. "The combination of drugs does not necessarily reduce cardiovascular events and stroke in an AF patient population and likely increases the risk of bleeding," he said.

"Even the most knowledgeable physicians may find themselves in a bind when encountering a patient who needs antiplatelet drugs, such as aspirin, due to coronary disease and blood thinners for atrial fibrillation," said senior author Gregory Marcus, MD, cardiologist and endowed professor in AF research at UC San Francisco School of Medicine. "While a large proportion of AF patients meeting the guidelines for stroke prevention medications fail to receive them, a lack of sufficient data regarding the clinical benefit among those with strong indications for both antiplatelet drugs and anticoagulants may in part be to blame."

Both Hsu and Marcus say concerns for bleeding may be the biggest reason for the underutilization of appropriate blood thinners in AF patients. However, the perception that aspirin by itself is sufficient or that the risk of aspirin plus a blood thinner is not worth the benefit may also be driving forces. Both researchers said more studies evaluating cardiovascular outcomes in AF patients prescribed aspirin-only versus oral blood thinners (or in combination) are needed.

Co-authors include: Thomas M. Maddox and Kevin Kennedy, VA Eastern Colorado Health Care System and University of Colorado School of Medicine; David F. Katz and Lucas N. Marzec, University of Colorado School of Medicine; Steven A. Lubitz, Cardiac Arrhythmia Service and Cardiovascular Research Center, Massachusetts General Hospital; Anil K. Gehi, and ll Mintu P. Turakhia, University of North Carolina.

This research was funded, in part, by the American College of Cardiology National Cardiovascular Data Registry.

http://www.eurekalert.org/pub_releases/2016-06/mc-mcs062016.php

Mayo Clinic study shows increase in Parkinson's disease over 30 years

First study to suggest an increasing trend in Parkinson's disease

ROCHESTER, Minn. -- The incidence of Parkinson's disease and parkinsonism increased significantly in 30 years from 1976 to 2005, Mayo Clinic researchers reported today in a study in JAMA Neurology. This trend was noted in particular for men age 70 and older. According to the researchers, this is the first study to suggest such an increasing trend. The study shows that men of all ages had a 17 percent higher risk of developing parkinsonism and 24 percent higher risk of developing Parkinson's disease for every 10 calendar years.

The study also showed that men 70 and older had an even greater increase -- a 24 percent higher risk of developing parkinsonism and 35 percent higher risk of developing Parkinson's disease for every 10 calendar years.

Using the Rochester Epidemiology Project, Mayo Clinic researchers were able to look at the complete medical records -- from birth to death -- of anyone in Olmsted County, Minnesota, who received at least one of the diagnoses related to parkinsonism. The records were reviewed by a movement disorders specialist to confirm the diagnosis and to classify different types of parkinsonism, including the most common type, Parkinson's disease.

"We have reasons to believe that this is a real trend," says Rodolfo Savica, M.D., Ph.D., lead author and neurologist at Mayo Clinic. "The trend is probably not caused merely by changes in people's awareness or changes in medical practice over time. We have evidence to suggest that there has been a genuine increase in the risk of Parkinson's disease. "The researchers point to environmental and lifestyle changes as potential causes for the increase.

"There has been a dramatic change in exposure to some risk factors in the United States," Dr. Savica says. "We know that environmental agents like pesticides or smoking or other agents in the environment have changed in the last 70 years or so. Changes in exposure to a number of risk factors may have caused Parkinson's disease to rise."

The study, based on almost 1,000 patients affected by parkinsonism, is the first to consider long-term trends in risk over 30 years. It also provides evidence contrary to two previous U.S. studies and one Canadian study that showed no trend, and particularly contrary to three United Kingdom studies that suggested a possible decline in the occurrence of Parkinson's disease over time.

The Mayo Clinic study also revealed a possible higher incidence of both parkinsonism and Parkinson's disease in men and women born from 1915 to 1924. "This observation is important because the persons born in that particular decade may have been exposed to some environmental or other factors during their intrauterine life or early after birth that increased the risk," Dr. Savica says. "We need to confirm this hypothesis.

"Parkinsonism is the umbrella term that includes Parkinson's disease but also may include other disorders. The diagnosis of parkinsonism requires the presence of slowness of movement and at least one other symptom -- a tremor while at rest, muscle rigidity or a tendency to fall. Parkinson's disease is defined as having the manifestations of parkinsonism but without any other known causes, and it is the most common type of parkinsonism.

The researchers urged caution in interpreting the trends, which may be from an increased awareness of symptoms and improved access to care. In the study's earlier years, for example, patients with cancer or severe cardiac disease may not have been diagnosed with parkinsonism or Parkinson's disease if doctors did not consider their movement disorder to be important in their care.

"Parkinson's disease is an important disease and a cause of disability, especially in older ages, and we don't want to have people untreated for a condition that is treatable just because they have four or five other diseases that are more prominent," Dr. Savica says.

The observation that the time trends were more evident in men than in women may support a genuine trend in incidence. Recognition of symptoms in the context of multiple illnesses should have changed similarly over time in men and women, the study notes. Thus, if the trend was not genuine it should have been similar in men and women.

Parkinsonism and Parkinson's disease tend to affect more men than women in general. But Dr. Savica also notes that the increase was more dramatic in men, but the study also showed a similar trend in women -- an increase in Parkinson's disease in women 70 years of age and older. However, the trend in women did not reach statistical significance.

"Differences in men and women may be important in understanding the environmental causes of Parkinson's disease," Dr. Savica says.

If the trend of increasing incidence rates is genuine, and can be replicated in other populations, it has major implications for finding the causes of Parkinson's disease and for public health, the researchers note. From a research perspective, the trend should prompt studies to identify environmental or lifestyle changes during the study subjects' lifespan. Environmental or lifestyle factors could include smoking, pesticide use, head trauma, coffee consumption and other factors.

Study co-authors are Brandon Grossardt, M.S.; James Bower, M.D.; Eric Ahlskog, M.D., Ph.D.; and senior author Walter Rocca, M.D., M.P.H., all of Mayo Clinic.

The study was supported by an award from the National Institute on Aging of the National Institutes of Health (grant AG 034676) and by the Mayo Foundation for Medical Education and Research.

http://www.eurekalert.org/pub_releases/2016-06/cmu-tsr062016.php

Ten simple rules to use statistics effectively

Under growing pressure to report accurate findings as they interpret increasingly larger amounts of data, researchers are finding it more important than ever to follow sound statistical practices.

For that reason, a team of statisticians including Carnegie Mellon University's Robert E. Kass wrote "Ten Simple Rules for Effective Statistical Practice." Published in *PLOS Computational Biology* for the journal's popular "Ten Simple Rules" series, the guidelines are designed to help the research community --

particularly scientists who aren't statistical experts or without a dedicated statistician as part of their team -- understand how to avoid the pitfalls of well-intended, but inaccurate statistical reasoning.

"A central and common task for us as research investigators is to decipher what data are able to say about the problems we are trying to solve," wrote Kass, professor of statistics and machine learning and interim co-director of the Center for the Neural Basis of Cognition, and his co-authors. "Statistics is a language constructed to assist this process, with probability as its grammar."

They continued, "While rudimentary conversations are possible without good command of the language (and are conducted routinely), principled statistical analysis is critical in grappling with many subtle phenomena to ensure that nothing serious will be lost in translation and to increase the likelihood that your research findings will stand the test of time."

The rules, which were made available online June 9, have received an extraordinary amount of attention so far with more than 37,000 page views, already making it one of the top 20 most viewed papers in the series, which includes about 60 total papers.. Their popularity doesn't surprise Michael J. Tarr, head of CMU's Department of Psychology.

"The sciences, and, particular the fields of psychology and neuroscience, have come under increasing scrutiny in recent years for sometimes poor statistical practices," Tarr said. "Straightforward and understandable guidelines as articulated by Kass and colleagues will help tremendously in reminding both students and faculty as to the importance of statistically well-grounded research. Their paper is an instant 'must-read' for anyone who cares about good and reproducible science."

A summary of the 10 rules:

#1 - Statistical Methods Should Enable Data to Answer Scientific Questions

Collaborating with statisticians is often most helpful early in an investigation because inexperienced users of statistics often focus on which technique to use to analyze data, rather than considering all of the ways the data may answer the underlying scientific question.

#2 - Signals Always Come With Noise

Variability comes in many forms, but it is crucial to understand when it is good and when it is noise in order to express uncertainty. It also helps to identify likely sources of systematic error.

#3 - Plan Ahead, Really Ahead

Asking questions at the design stage can save headaches at the analysis stage. Careful data collection also can greatly simplify analysis and make it more rigorous.

#4 - Worry About Data Quality

When it comes to data analysis, "garbage in produces garbage out." The complexity of modern data collection requires many assumptions about the function of technology, often including data pre-processing technology, which can have profound effects that can easily go unnoticed.

#5 - Statistical Analysis Is More Than a Set of Computations

Statistical software provides tools to assist analysis, not define them. The scientific context is critical, and the key to principled statistical analysis is to bring analytical methods into close correspondence with scientific questions.

#6 - Keep it Simple

Simplicity trumps complexity. Large numbers of measurements, interactions among explanatory variables, nonlinear mechanisms of action, missing data, confounding, sampling biases and other factors can require an increase in model complexity.

But, keep in mind that a good design, implemented well, can often allow simple methods of analysis to produce strong results.

#7 - Provide Assessments of Variability

A basic purpose of statistical analysis is to help assess uncertainty, often in the form of a standard error or confidence interval, and one of the great successes of statistical modeling and inference is that it can provide estimates of standard errors from the same data that produce estimates of the quantity of interest. When reporting results, it is essential to supply some notion of statistical uncertainty.

#8 - Check Your Assumptions

Widely available statistical software makes it easy to perform analyses without careful attention to inherent assumptions, and this risks inaccurate, or even misleading, results. It is therefore important to understand the assumptions embodied in the methods and to do whatever possible to understand and assess those assumptions.

#9 - When Possible, Replicate!

Ideally, replication is performed by an independent investigator. The scientific results that stand the test of time are those that get confirmed across a variety of different, but closely related, situations. In many contexts, complete replication is very difficult or impossible, as in large-scale experiments such as multi-center clinical trials. In those cases, a minimum standard would be to follow Rule 10.

#10 - Make Your Analysis Reproducible

Given the same set of data, together with a complete description of the analysis, it should be possible to reproduce the tables, figures and statistical inferences. Dramatically improve the ability to reproduce findings by being very systematic

about the steps in the analysis, by sharing the data and code used to produce the results and by following accepted statistics best practices.

In addition to Kass, the co-authors are Johns Hopkins University's Brian S. Caffo, North Caroline State University's Marie Davidian, Harvard University's Xiao-Li Meng, and Nancy Reid of the University of California Berkeley and the University of Toronto.

"I am a big believer in the value of identifying major ideas in statistics, and stating them clearly and concisely," Kass said. "The 10 simple rules series is terrific, having proven its worth as a format for high-level scientific concepts. This article was pretty hard work, but we had a great team and I was extremely happy with the result."

http://journals.plos.org/ploscompbiol/article?id=10.1371/journal.pcbi.1004961.

http://www.eurekalert.org/pub_releases/2016-06/mu-emb062016.php

Exercise may be the simple solution for rescuing seniors' lost and injured muscle

Exercise may have some surprising benefits for seniors who experience rapid muscle loss and muscle injury and loss as they age.

Researchers at McMaster University have found that physical activity can help retain, even repair and regenerate damaged muscle in the elderly. The findings challenge what is generally seen as an inevitable fact of life: that muscle atrophy and damage cannot be completely repaired in old age and in some cases lost altogether.

Researchers compared and analyzed the capacity for muscle repair performance ofin a group of young mice, a group of old sedentary mice and a group of old exercise-trained mice. three groups of young and old mice, some of which had experienced muscle injury, some of which had been exercise trained, and others which had not.

"The world's older population is rapidly growing and preventing muscle loss and promoting muscle repair is paramount to preserve health," says Gianni Parise, lead author of the study and an associate professor in the Department of Kinesiology at McMaster. "These findings suggest that age-related compromised muscle repair can be rescued with regular exercise," he says.

After eight weeks of exercise, researchers found the old mice were able to repair and rebuild muscle more quickly following injury when compared to the old mice which had not exercised. And after a period of 28 days, muscle repair was comparable to that of young mice. Old mice that had not exercised did not fully recover.

The findings suggest that exercise can be used as a preventative measure in older adults who lose muscle rapidly when their activity levels fall, which can happen for a variety of reasons, including illness or extended hospital stays. "Quite simply, this demonstrates the importance of remaining active throughout life," says Parise. "Regular exercise can preserve basic processes that govern muscle health."

The research was published in the FASEB Journal.

http://bit.ly/28UoV50

Brain Tumor Risk Linked with Higher Education, Study Finds

People with higher levels of education may be more likely to develop certain types of brain tumors, a new study from Sweden suggests.

By Agata Blaszczak-Boxe, Contributing Writer

Researchers found that women who completed at least three years of university courses were 23 percent more likely to develop a type of cancerous brain tumor called glioma, compared with women who only completed up to nine years of mandatory education and did not go to a university. And men who completed at least three years of university courses were 19 percent more likely to develop the same type of tumor, compared with men who did not go to a university.

Though the reasons behind the link are not clear, "one possible explanation is that highly educated people may be more aware of symptoms and seek medical care earlier," and therefore are more likely to be diagnosed, said Amal Khanolkar, a research associate at the Institute of Child Health at the University College Londonand a co-author of the study.

In the study, the researchers looked at data on more than 4.3 million people in Sweden who were a part of the Swedish Total Population Register. The researchers tracked the people for 17 years, beginning in 1993, to see if they developed brain tumors during that time. They also collected information about the people's education levels, income, marital status and occupation.

During the 17-year study, 5,735 men and 7,101 women developed brain tumors, according to the findings, published today (June 20) in the Journal of Epidemiology & Community Health.

In addition to the differences between brain tumor development and education level, the researchers also found an association between brain tumor development and income. Men who had higher incomes were 14 percent more likely to develop glioma during the study period, compared with men with lower incomes, according to the study. However, the relationship between the risk of this type of brain tumor and income level was not found in women, the researchers said.

Moreover, the study found that men who worked in managerial and professional roles were 20 percent more likely to develop glioma, compared with those who worked manual jobs. These men were also 50 percent more likely to develop acoustic neuroma, a type of noncancerous brain tumor that grows on the nerve that is used for hearing and balance.

The researchers also found that women who completed at least three years of university courses were also 16 percent more likely to develop a type of mostly noncancerous brain tumor called meningioma, compared with women who did not go on to higher education.

The new findings are in line with previous research, which has also found a link between an increased risk of certain brain tumors and higher socio-economic status, the researchers said.

However, the new study only shows a link between certain types of brain tumor and certain factors; it does not show that these factors directly cause brain tumors, the researchers said. While other lifestyle factors might have also played a role, the researchers did not have access to such information, they said.

The idea that education levels and brain tumors are linked is not entirely new.

"It has been an 'urban legend' among neurosurgeons that smarter people are more likely to get brain tumors," said Dr. Raj K. Narayan, the chair of neurosurgery at North Shore University Hospital in Manhasset, New York, who was not involved in the new study. "However, I am somewhat surprised to find that this may actually be true." The mechanism behind this link is still unknown, but it might be that having more brain cells or greater brain activity somehow increases a person's risk of brain tumors, he told Live Science.

http://bit.ly/28RMQ3L

E.T. Phones Earth? 1,500 Years Until Contact, Experts Estimate

E.T. Phones Earth? 1,500 Years Until Contact, Experts Estimate

By Nola Taylor Redd, Live Science Contributor | June 20, 2016 12:02pm ET

"Communicating with anybody is an incredibly slow, long-duration endeavor," said Evan Solomonides at a press conference June 14 at the American Astronomical Society's summer meeting in San Diego, California. Solomonides is an undergraduate student at Cornell University in New York, where he worked with Cornell radio astronomer Yervant Terzian to explore the mystery of the Fermi paradox: If life is abundant in the universe, the argument goes, it should have contacted Earth, yet there's no definitive sign of such an interaction.

Solomonides said the enormous size of the galaxy means the silence comes as no surprise.

"Space is very big. It takes a long time to reach anyone, even at the speed of light," he said.

Silent space

When Enrico Fermi formulated his namesake paradox in the 1950s, planets around other stars were only hypothetical. Today, scientists suspect that nearly every sun has at least one if not more worlds, dramatically increasing the chances for life to have evolved throughout the universe. However, for some people, the lack of confirmed greeting from another civilization suggests that life may not be so common after all.

Solomonides applied the mediocrity principle — the idea that Earth's attributes are likely common in the rest of the universe, rather than unusual — to the Fermi paradox. Scientists think that Earth is an average planet around an average star, orbiting in an average place within an average galaxy.

"There's nothing even remotely special about where we are in the universe, or even in the galaxy," Solomonides said.

The 1936 Berlin Olympic games broadcast was the first radio signal strong enough to leave Earth. Traveling at the speed of light, this program is the leading edge of a bubble of broadcasts racing outward through space from Earth. But that signal has managed to travel only 80 light-years from the planet.

Solomonides said advanced life elsewhere in the universe is unlikely to have arisen much before life on Earth. That's because bodies like those of humans require a mixture of the heavy elements produced over the lifetimes of stars, and it takes several generations of star formation to produce the necessary quantities. As a result, civilizations capable of communicating throughout the galaxy wouldn't have started out much earlier than happened on Earth.

Based on the assumption that life and technology on Earth should have evolved at a relatively average pace, not significantly faster or slower than for other civilizations, Solomonides calculated the communication bubbles that life would produce throughout the galaxy. He found that, as of today, only about a tenth of 1 percent of the Milky Way would be blanketed by signals. With those numbers, it's likely that Earth won't hear from other life-forms for another 1,500 years.

"There could be life everywhere in the galaxy, and we still wouldn't know it," Solomonides said. In fact, "if we had been contacted by another civilization, we would actually be special."

That doesn't mean humanity should stop searching for signals or cut off broadcasts (though the rise of cable to carry television signals may mean that Earth is getting quieter anyway), Solomonides said. Instead, humans should keep broadcasting and listening in order to avoid missing the historic chance of contact, he said. People simply shouldn't expect results any time in the near future.

Even if, in the next 2,000 years, humans still haven't heard from other life-forms, that won't mean life doesn't exist throughout the galaxy, Solomonides said. He pointed out that communication requires the evolution of advanced life; molecular life won't be sending out signals, and so isn't considered in the search for extraterrestrial intelligence. Other scientists have suggested alien life may have evolved but then died out.

Another possibility is that advanced civilizations are unwilling to respond, because they'd rather avoid contact. After all, Solomnides said, Earth's first broadcast was Adolf Hitler's Olympics remarks.

"That's not really a great introduction," he said, wryly.

http://www.bbc.com/news/world-africa-36548837

Angola's front line against yellow fever

More than 300 people have so far died in the current yellow fever outbreak in Angola

By Firle Davies BBC News, Angola

In the green and shaded gardens of the Americo Boavida Hospital in Angola's capital, Luanda, women in colourful printed dresses wait patiently for visiting hours to begin. It is one of the biggest hospitals in the city, serving almost two million people. Malaria is the most common killer here, but since December last year they have had to counter another, potentially more dangerous, mosquito-borne virus: Yellow fever.

In its 16 June report, the World Health Organization (WHO) said that 345 people are reported to have died from yellow fever in the last seven months among more than 3,000 cases in Angola. Not since 1971 has there been such a serious outbreak, and the reasons why it has happened now are complex and many.

Dr Fortunato Silva, the clinical director at Americo Boavida, says that this outbreak is more worrying not only in relation to the number of cases, but also the number of deaths. He thinks some of the reasons for this may include the virus becoming more virulent, immunity levels amongst the population dropping and most critically, people not vaccinating as they should.

"Ninety patients have been hospitalised with fever, jaundice and haemorrhaging, since 23 February," Dr Silva said. "There have been 33 deaths, which is a very high mortality rate. All of them had tested negative for malaria."

Angola's health system is well regarded, and there are established countrywide vaccination and awareness programmes. Since 1989, babies have been vaccinated against yellow fever at the age of nine months, and children cannot attend school unless they have a valid yellow fever certificate.

Despite this, Dr Silva says, something is not working, and there are questions that need to be answered in terms of public health strategies.

Dr Francisco Songane, the representative in Angola for the UN children's fund, Unicef, describes what is happening as "a major crisis". He says critical time was lost between samples being taken, tests being run, and results finally arriving three weeks later. By the time confirmation of yellow fever came, it had spread from the densely populated area of K30, part of the capital's Viana district, and then across the entire city.

The spread of yellow fever

Angola - 345 reported deaths, 3,137 suspected cases
Democratic Republic of the Congo - 71 reported deaths, 1,044 suspected cases (not all linked to Angola)
Kenya - two suspected cases (travellers returning from Angola)
China - 11 suspected cases (travellers returning from Angola)
Source: WHO

Dr Songane says that though measures were immediately put in place, in a city such as Luanda, where there are "huge neighbourhoods... a sea of so many houses, so populated, the virus spread very quickly".

And to compound the already serious outbreak, there was a global shortage of the vaccine to contend with. And vaccines must be administered within 10 days of the outbreak being identified. "The timeline was not being met," he says. "And the shortage of vaccine meant that by then it had spread beyond Luanda."

The shortage now means that the WHO now recommends cutting the standard dose of yellow fever vaccine by 80%. The smaller dose would provide immunity for at least 12 months, it says.

Further adding to the crisis was the widespread use of fake yellow fever certificates, because, as Dr Songane explains "there were also misconceptions that the vaccine will kill you, that you will get a disease. "There is a need to counter this and get the message to the people that you can do good for yourself, your community, your neighbourhoods. "Fake certificates will not protect you. That's the message."

Nearly all of Luanda's population has now been vaccinated, and the ministry of health is in a race against time to get enough of the vaccine manufactured for the rest of the country.

What is yellow fever?

Caused by a virus that is transmitted to humans by mosquitoes
Difficult to diagnose and often confused with other diseases or fevers
Presence of yellow fever antibodies can be detected by blood tests
Most people recover after the first phase of infection that usually involves fever, muscle and back pain, headache, shivers, loss of appetite, and nausea or vomiting
About 15% of people face a second, more serious phase involving high fever, jaundice, bleeding and deteriorating kidney function
Half of those who enter the "toxic" phase usually die within 10 to 14 days The rest recover

Source: WHO

Angola strictly enforces international regulations on travellers coming in and out of the country, and Dr Songane says that whilst hard lessons have been learned, every country must now enforce these regulations.

Vaccines take six months to produce and if there is another serious outbreak, global supplies will not be able to keep up with the demand.

But it is too late to prevent its spread beyond Angola's borders.

Neighbouring Democratic Republic of Congo has declared a localised yellow fever epidemic in three provinces, including the capital Kinshasa. Cases related to the outbreak in Angola have also surfaced in Kenya and China.

The mosquito that carries the virus, aedes aegypti, is found in most of the country, but it is prevalent in Luanda, made worse now by uncollected refuse.

Angola is one of Africa's largest oil producers, and nearly 70% of the government's income derives from oil. In the boom years the country invested heavily in infrastructure projects and social welfare, but now with the collapse in the price of oil, basic services like garbage collection have stopped.

Rotting rubbish can be see piling high along with sewage running through crowded neighbourhood streets.

Dr Songane argues that it is urgent that a way is found to solve the issue of the rubbish piling up. "People cannot continue to live in these conditions," he says.

"This has become a rich and beautiful place for this mosquito to bite, and it's made worse because it bites during the day. [The rubbish] is in the streets and people are being exposed every day."

Only some of those who carry the yellow fever virus get the symptoms, but the rest, who may be unaware that they are carriers, can be bitten by the mosquito and therefore help spread the virus.

Dr Songane says the outbreak is not yet under control. He is aware that much needs to be done, but there is no sense of panic. It is only when transmissions stop, he says, "that we can breathe. It's not over until it's over".

http://bit.ly/28S0GjM

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

Debora MacKenzie highlights the threats to civilisation – and suggests solutions

By Debora MacKenzie

It’s getting scary. On Monday a yellow fever epidemic was declared in Kinshasa, the sprawling capital of the Democratic Republic of the Congo. The disease spread there from Angola, where it has been circulating since an outbreak began in December. There have been over 4000 known or suspected cases in the two countries, and the virus is thought to kill in some 5 per cent of cases.

In an unprecedented move, last week the World Health Organization announced that experimental evidence shows the vaccine can be diluted fivefold to make stocks stretch further, in a bid to control the outbreak in Africa.

Before this outbreak, Angola was not considered at risk so few people there had been vaccinated. Since February in Angola, and since March in Congo, 18 million doses of vaccine have been given to try and stop the virus spreading. There is not enough vaccine left to contain a major epidemic in Kinshasa without eating into stocks meant for child vaccinations, says Seth Berkley, head of GAVI, the international vaccine agency.

It should never have come to this. Control of the mosquitoes that carry yellow fever drove the virus out of countries such as the US, where it used to cause deadly epidemics in places like Philadelphia. The vaccine for yellow fever, introduced in 1936, is cheap, safe and possibly the world’s most effective: one dose protects a person for life. By the 1970s mosquito control and vaccination had cut yellow fever to very low levels across tropical and subtropical Africa and South America.

Eye off the ball

But “success caused failure”, says Duane Gubler of Duke-NUS Medical School in Singapore. As the disease disappeared countries stopped vaccinating, and financial crises plus rocketing urban populations hampered mosquito control.

The virus cannot be eradicated from its tropical strongholds, as it normally lives harmlessly in forest-dwelling monkeys and the mosquitoes that bite them. But when a person infected by a forest mosquito travels to town, urban Aedes mosquitoes can pick up and spread the virus, causing an explosive outbreak when densities of susceptible people and mosquitoes are high.

Aedes mosquitoes are the very ones that spread dengue, chikungunya and most recently Zika virus. “The rapid international spread of those viruses shows why we are worried about yellow fever,” says Gubler.

An increase in urban outbreaks of yellow fever since the 1980s led to resumed vaccination of children in 2005 in West African countries most at risk, and a stockpile of 6 million doses of vaccine managed by the WHO. But “marketing considerations did not make it attractive for the manufacturers to make more than the usual stockpile”, says Jack Woodall of the Federal University of Rio de Janeiro, Brazil.

That is why the world is out of vaccine. The stockpile was used up, then refilled and used up twice again, with vaccine procured from supply chains worldwide to battle the epidemic in Africa. More vaccine cannot be made quickly as it is grown in germ-free chicken eggs, which are scarce.

Difficult to spot

Meanwhile, some 1.8 billion people in Asia live with Aedes mosquitoes, so would be vulnerable if yellow fever, carried by migrant workers in Africa for instance, took hold there. China has already detected the virus in 11 workers returning from Angola, fortunately to homes north of the country’s Aedes belt.

Other countries may not detect the virus until it has spread. “Delhi has so much hepatitis that a fever with jaundice would never be suspected to be yellow fever,” says Woodall, and India’s diagnostic labs are not equipped to pick it up.

Gubler hopes Asia’s ongoing dengue epidemic might help. Yellow fever is a related virus, and animal experiments in the 1970s suggested dengue antibodies might partially protect people from yellow fever, and perhaps slow its spread. Woodall, however, fears yellow fever could behave like many viral diseases when they invade new territory, with even higher death rates than in its native range.

Right now, both are hoping we don’t have to find out. “Some of us have been warning for years that the question wasn’t whether this could happen, but when,” says Gubler. Now the world is facing an unknown, potentially severe risk from a disease we had under control in the 1960s.

http://bit.ly/28UxUmS

Farming Invented Twice in the Middle East, Genomes Study Reveals

A study of 44 ancient genomes supports the idea of independent farming revolutions in the Fertile Crescent

By Ewen Callaway, Nature magazine on June 21, 2016

Two Middle Eastern populations independently developed farming and then spread the technology to Europe, Africa and Asia, according to the genomes of 44 people who lived thousands of years ago in present-day Armenia, Turkey, Israel, Jordan and Iran.

Posted on June 17 on the bioRxiv preprint server, the research supports archaeological evidence about the multiple origins of farming, and represents the first detailed look at the ancestry of the individuals behind one of the most important periods in human history—the Neolithic revolution.

Some 11,000 years ago, humans living in the ancient Middle East region called the Fertile Crescent shifted from a nomadic existence, based on hunting game and gathering wild plants, to a more sedentary lifestyle that would later give rise to permanent settlements. Over thousands of years, these early farmers domesticated the first crops and transformed sheep, wild boars and other creatures into domestic animals.

Dozens of studies have examined the genetics of the first European farmers, who emigrated from the Middle East beginning some 8,000 years ago, but the hot climes of the Fertile Crescent had made it difficult to obtain ancient DNA from remains found there. Advances in extracting DNA from a tiny ear bone called the petrous allowed a team led by Iosif Lazaridis and David Reich, population geneticists at Harvard Medical School in Boston, Massachusetts, to analyse the genomes of the 44 Middle Eastern individuals, who lived between 14,000 and 3,500 years ago.

Geographical divide

The team found stark differences between the genomes of Neolithic individuals from the southern Levant region, including Israel and Jordan, and those living across the Zagros Mountains in western Iran. The Zagros early farmers were instead more closely related to nearby hunter-gatherers who lived in the region before the Neolithic.

This pattern of ancestry adds to the evidence that the hunter-gatherers in the southern Levant and Iran independently developed farming, says Roger Matthews, an archaeologist at the University of Reading, UK, who co-directs the Central Zagros Archaeological Project in Iran.

“There has been a school of thought arguing that everything happens first in the southern Levant and everyone learns how to be farmers from this initial dispersal,” he says. “But the archaeological evidence shows very strong local traditions that are clearly not in communication with each other, persisting for centuries if not millennia.”

The Zagros farmers domesticated goats as well as cereals such as emmer, whereas their counterparts to the west had their own crops, including barley and wheat. Around 9,500 years ago, these traditions began spreading around the Middle East, Rogers says, noting that the two populations of farmers may have mixed in

eastern Turkey while seeking out sources of obsidian, which was useful for making tools. By the time farmers in present-day Turkey began migrating to Europe, they carried a 'Neolithic toolkit' that included crops, animals and tools from both farming traditions.

How farming fanned out

The latest study also finds traces of the diverse foundations of farming beyond Europe. Iranian farmers moved north into the Eurasian steppe and eastwards into present-day India and Pakistan. Southern Levant farmers made a trek to Africa, perhaps bringing new farming traditions to East Africa. The study notes that this is in line with previous work suggesting that humans from Eurasia launched a 'back-to-Africa' migration some 3,000 years ago.

Rogers says that the study offers a first glimpse of the spread of farming to Asia, "but we need an awful lot more work looking at how farming spread eastward," he says.

"The study has the grandeur of providing a big picture of the advent of farming and how this shift from hunting and gathering to food production had a crucial impact on the evolutionary history of Europe, East Africa, India and Central Asia," says Carles LaLueza-Fox, a palaeogeneticist at the Institute of Evolutionary Biology in Barcelona, Spain.

A second bioRxiv preprint, posted by some of the same team on June 19, reports the whole genome of one the Zagros farmers: a female who lived 10,000 years ago. (By contrast, the report from Reich and Lazaridis samples thousands of single-letter genetic variants peppered across the genomes of the 44 individuals). Differences between her genome and those of farmers in Turkey supports the broad conclusion that farming emerged independently in the region, the paper says.

LaLueza-Fox sees such research as an indication that scientists can reliably collect ancient DNA from hotter climates, where much of human prehistory played out. "Retrieving genomic data from the ancient Near East is a palaeogenomic dream come true," he says.

http://www.eurekalert.org/pub_releases/2016-06/cp-ifm061416.php

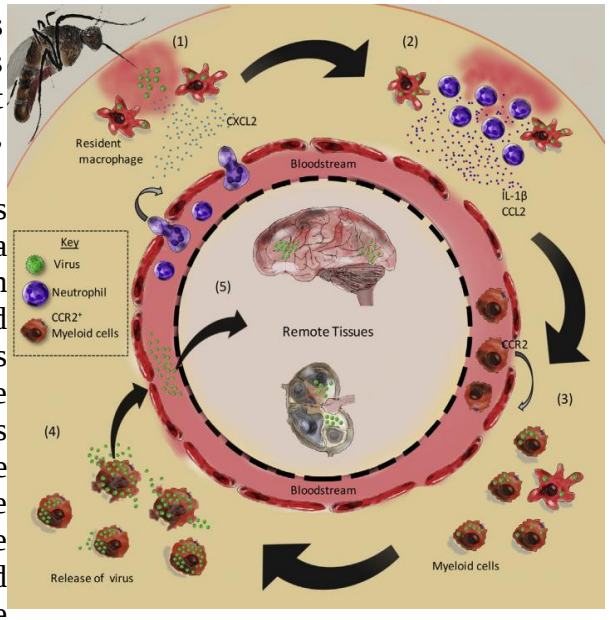
Inflammation from mosquito bites may enhance viral infection

The itchy, red welts that appear after being bitten by a mosquito may help any viruses the insect is carrying pass on to a new host.

A mouse study published June 21 in Immunity suggests that the swelling and irritation that make mosquito bites so unpleasant may provide a mechanism by which viruses like Zika are able to replicate and spread.

"Before we did this study, little was known about the events and processes that occur at mosquito bite sites," says Clive McKimmie, a Research Fellow at the University of Leeds and the paper's senior author. "Our findings suggest that the inflammatory response at these sites helps viruses to replicate, enhancing their ability to cause disease."

In the new research, the investigators used mouse models to study the bites of the Aedes aegypti mosquito, the species that spreads infections such as Zika, dengue, and chikungunya.



When a mosquito bites, it injects saliva into the skin. The saliva triggers an immune response, in which white blood cells called neutrophils and myeloid cells rush to the site. In this study, the team injected mice with viruses into the skin with or without the presence of a mosquito bite at the injection site and compared the reaction. They found that instead of helping, some of these immune cells get infected and inadvertently replicate virus.

The inoculation of viruses into mosquito bite sites is an important and common stage of arbovirus infections. This visual abstract depicts the findings of McKimmie and colleagues, who show that inflammation at bite sites aids viral replication and dissemination in vivo, resulting in more severe infection. These findings define additional targets for post-exposure prophylactic intervention. Pingen et al./Immunity 2016

In the absence of mosquito bites and their accompanying inflammation, the viruses failed to replicate well. But the presence of mosquito bites at the infection site resulted in an order of magnitude higher levels of virus. Further studies showed that the influx of white blood cells was required for enhanced replication of the viruses.

According to McKimmie, previous studies that have used in vivo models to study the course of mosquito-borne infections haven't looked at the bite as a necessary component. "We think the bite itself is affecting the systemic course and clinical outcome of the infection," he says. "If you want an in vivo model that replicates the most relevant parts of infection, you should include this inflammatory aspect."

"This was a big surprise we didn't expect," he adds. "These viruses are not known for infecting immune cells. And sure enough, when we stopped these immune cells from coming in, the bite did not enhance the infection anymore."

Although this research is still early work done in mice, McKimmie says the finding suggests new approaches for combating viruses that lead to health problems in humans. "We're quite keen to see if using topical creams to suppress bite inflammation will enable you to stop a virus from making someone as sick as it otherwise would do," he says.

He notes that if it's proven effective, this approach could work against future virus outbreaks that we don't know about yet. "Nobody expected Zika, and before that nobody expected chikungunya," he says. "There are estimated to be hundreds of other mosquito-borne viruses out there and it's hard to predict what's going to start the next outbreak."

This research was supported by the UK Medical Research Council and the UK Biotechnology and Biological Sciences Research Council.

Immunity, Pingen et al: "Host inflammatory response to mosquito bites enhances severity of arbovirus infection." http://www.cell.com/immunity/fulltext/S1074-7613(16)30205-9

http://www.eurekalert.org/pub_releases/2016-06/bu-rbc062116.php

Research bolsters case for a present-day subsurface ocean on Pluto

When the NASA's New Horizons spacecraft buzzed by Pluto last year, it revealed tantalizing clues that the dwarf planet might have -- or had at one time -- a liquid ocean sloshing around under its icy crust

PROVIDENCE, R.I. [Brown University] -- According to a new analysis led by a Brown University Ph.D. student, such an ocean likely still exists today.

The study, which used a thermal evolution model for Pluto updated with data from New Horizons, found that if Pluto's ocean had frozen into oblivion millions or billions of years ago, it would have caused the entire planet to shrink. But there are no signs of a global contraction to be found on Pluto's surface. On the contrary, New Horizons showed signs that Pluto has been expanding.

"Thanks to the incredible data returned by New Horizons, we were able to observe tectonic features on Pluto's surface, update our thermal evolution model with new data and infer that Pluto most likely has a subsurface ocean today," said Noah Hammond, a graduate student in Brown's Department of Earth, Environmental and Planetary Sciences, and the study's lead author.

The research, which Hammond coauthored with advisors Amy Barr of the Planetary Science Institute in Arizona and Brown University geologist Marc Parmentier, is in press in Geophysical Research Letters.

The pictures New Horizons sent back from its close encounter with the Kuiper Belt's most famous denizen showed that Pluto was much more than a simple snowball in space. It has an exotic surface made from different types of ices -- water, nitrogen and methane. It has mountains hundreds of meters high and a vast heart-shaped plain. It also has giant tectonic features -- sinuous faults hundreds of kilometers long as deep as 4 kilometers. It was those tectonic features that got scientists thinking that a subsurface ocean was a real possibility for Pluto.

"What New Horizons showed was that there are extensional tectonic features, which indicate that Pluto underwent a period of global expansion," Hammond said. "A subsurface ocean that was slowly freezing over would cause this kind of expansion."

Scientists think that there may have been enough heat-producing radioactive elements within Pluto's rocky core to melt part of the planet's ice shell. Over time in the frigid Kuiper belt, that melted portion would eventually start to refreeze. Ice is less dense than water, so when it freezes, it expands. If Pluto had on ocean that was frozen or in the process of freezing, extensional tectonics on the surface would result, and that's what New Horizons saw.

There aren't many other ways on Pluto to get such features. One way might have been through a gravitational tug of war with its moon, Charon. But the active gravitational dynamics between the two have long since wound down, and some of the tectonics look fairly fresh (on a geologic timescale). So, many scientists believe that an ocean is the strongest scenario.

But if Pluto had an ocean, what is its fate today? Could the freezing process still be going on, or did the ocean freeze solid a billion years ago?

That's where the thermal evolution model run by Hammond and his colleagues comes in. The model includes updated data from New Horizons on Pluto's diameter and density, key parameters in understanding the dynamics in Pluto's interior. The model showed that because of the low temperatures and high pressure within Pluto, an ocean that had completely frozen over would quickly convert from the normal ice we all know to a different phase called ice II. Ice II has a more compact crystalline structure than standard ice, so an ocean frozen to ice II would occupy a smaller volume and lead to a global contraction on Pluto, rather than an expansion.

"We don't see the things on the surface we'd expect if there had been a global contraction," Hammond said. "So we conclude that ice II has not formed, and therefore that the ocean hasn't completely frozen."

There are a few caveats, the researchers point out. The formation of ice II is dependent on the thickness of Pluto's ice shell. Ice II only forms if the shell is 260 kilometers thick or more. If the shell is thinner than that, the ocean could have

frozen without forming ice II. And if that were the case the ocean could have frozen completely without causing contraction.

However, the researchers say there's good reason to believe that the ice shell is more than 260 kilometers. Their updated model suggests that Pluto's ice shell is actually closer to 300 or more kilometers thick. In addition, the nitrogen and methane ices that New Horizons found on the surface bolster the case for a thick ice shell. "Those exotic ices are actually good insulators," Hammond said. "They may be helping Pluto from losing more of its heat to space."

Taken together, the new model bolsters the case for an ocean environment in the furthest reaches of the solar system.

"That's amazing to me," Hammond said. "The possibility that you could have vast liquid water ocean habitats so far from the sun on Pluto -- and that the same could also be possible on other Kuiper belt objects as well -- is absolutely incredible."

The research was supported by the NASA Earth and Space Science Fellowship (NNX13AN99H) and NASA Planetary Geology & Geophysics (NNX15AN79G).

http://bit.ly/290fxwx

Hundreds of genes seen sparking to life two days after death

The discovery that many genes are still working up to 48 hours after death has implications for organ transplants, forensics and our very definition of death

By Anna Williams

When a doctor declares a person dead, some of their body may still be alive and kicking – at least for a day or two. New evidence in animals suggests that many genes go on working for up to 48 hours after the lights have gone out.

This hustle and bustle has been seen in mice and zebrafish, but there are hints that genes are also active for some time in deceased humans. This discovery could have implications for the safety of organ transplants as well as help pathologists pinpoint a time of death more precisely, perhaps to within minutes of the event.

Peter Noble and Alex Pozhitkov at the University of Washington, Seattle, and their colleagues investigated the activity of genes in the organs of mice and zebrafish immediately after death. They did this by measuring the amount of messenger RNA present. An increase in this mRNA – which genes use to tell cells to make products such as proteins – indicates that genes are more active.

Noble's team measured mRNA levels in zebrafish, and in brain and liver samples from mice at regular intervals for up to four days after death. They then compared these with mRNA levels measured at the time of death.

As you might expect, overall mRNA levels decreased over time. However, mRNA associated with 548 zebrafish genes and 515 mouse genes saw one or more peaks of activity after death. This meant there was sufficient energy and cellular function for some genes to be switched on and stay active long after the animal died.

These genes cycled through peaks and dips in activity in a "non-winding down" manner, unlike the chaotic behaviour of the rest of the decaying DNA, says Noble. Hundreds of genes with different functions "woke up" immediately after death. These included fetal development genes that usually turn off after birth, as well as genes that have previously been associated with cancer. Their activity peaked about 24 hours after death.

A similar process might occur in humans. Previous studies have shown that various genes, including those involved in contracting heart muscle and wound healing, were active more than 12 hours after death in humans who had died from multiple trauma, heart attack or suffocation (*Forensic Science International*, doi.org/bj63).

The fact that some genes associated with cancer are activated after death in animals, might be relevant for reducing the incidence of cancer in people who receive organ transplants, says Noble. People who get a new liver, for example, have more cancers after the treatment than you would expect if they hadn't had a transplant. The regime of drugs they need to take for life to suppress their immune system so it doesn't attack the new organ may contribute to this, but Noble says it is worth investigating if activated cancer genes in the donor liver could play a part.

So why do so many genes wake up after death? It is possible that many of the genes become active as part of physiological processes that aid healing or resuscitation after severe injury. For example, after death, some cells might have enough energy to kick-start genes involved in the inflammation process to protect against damage – just as they would if the body were alive.

Alternatively, a rapid decay of genes that normally suppress other genes – such as those involved in embryological development – might allow the usually quiet genes to become active for a short period of time.

For forensic scientists, knowing how gene activity rises and falls at different time points after death is useful for working out when someone died. Measuring mRNA would allow us to nail down the time since death to hours and possibly even minutes, rather than days, helping to reconstruct events surrounding the death.

It is good to see such progress being made in this area, says Graham Williams, consultant forensic geneticist at the University of Huddersfield, UK. "But substantial work is required before this could be applied to case work."

The research also raises important questions about our definition of death – normally accepted as the cessation of a heartbeat, brain activity and breathing. If

genes can be active up to 48 hours after death, is the person technically still alive at that point? “Clearly, studying death will provide new information on the biology of life,” says Noble.

Kiss of death

What happens when we die? Well, that depends on where we end up. A body that has been refrigerated and encased in a coffin could take decades to completely decompose.

But out in the open, the human body can disappear in just months. Here, within minutes of death, carbon dioxide starts to accumulate in our blood, causing cells to burst open and spew out enzymes that digest tissues. Within half an hour, blood starts to pool at the lowest point, while the rest of the body turns pale. Rigor mortis then sets in as calcium ions diffuse into cells causing muscles to contract.

Three days later, putrification occurs as microbes that live in our gut break down proteins, creating a repulsive odour. They produce gases that bloat the body, which after two weeks collapses.

Our flesh is rapidly consumed by bacteria and maggots. Eventually, after months or years, only bones are left – minus their collagen – which succumbs to bacteria and fungi.

Journal references: BioRxiv, DOI: 10.1101/058305; DOI: 10.1101/058370

http://www.bbc.com/news/health-36586665

Ginger and acupressure 'options for morning sickness'

Taking ginger or using acupressure on the wrist may help some women with mild morning sickness, the Royal College of Obstetricians and Gynaecologists (RCOG) says.

Its guidance suggests these therapies could offer alternatives to women who want to avoid medication. But it says anti-sickness drugs and hospital treatment are important in more severe cases. The recommendations are in line with advice from NHS watchdog NICE. Nausea and vomiting affects about 80% of pregnant women.

'Lack of understanding'

For many, it disappears within the first four months - though symptoms are not confined to the morning hours as its commonly used name suggests.

In its first guidance on the issue, the RCOG says treatment can vary around the UK and there is an occasional lack of understanding of the condition's severity.

Its guidelines weigh up the evidence for a range of treatments - including complementary therapies - and set out specific options depending on how severe the condition is. Anti-sickness drugs can help in the many cases, it says. And some women may need day visits to hospitals or longer admissions for fluids and medication.

Ginger biscuits

Meanwhile, for women with mild or moderate symptoms who do not want to use drugs, acupressure (wearing a special bracelet that applies pressure) may help.

The guidance also mentions studies showing that ginger can provide some relief - including one study using ginger biscuits. But NHS Choices warns that as ginger products are not licensed for medical use in the UK, supplements should be bought only from reputable sources. And anyone still experiencing problems should seek medical advice.

Meanwhile, the RCOG says hypnosis is not recommended as there is not enough evidence to establish whether it is effective.

For women with more difficult symptoms, including a very severe form, known as hyperemesis gravidarum (HG), specialist treatment, including hospital admission and mental health support, should be offered, it suggests.

Caitlin Dean, who chairs the Pregnancy Sickness Support charity and had HG in three pregnancies, said: "On top of the nausea and vomiting (this could be up to 30 times a day), I had a pounding headache, incredibly heightened sense of smell and excessive saliva.

"My days would be spent lying in bed with a quiet audio-book as I couldn't read or watch TV because it all made me sick. "I soon became very dehydrated and was admitted to hospital at eight weeks pregnant. "I was housebound for most of the pregnancy, which made me feel incredibly lonely."

Dr Manjeet Shehmar, lead author of the guidelines, said many women with persistent symptoms were not receiving the treatment they needed. "Women with persistent nausea can often feel that there is a lack of understanding of their condition," she said. "They may be unable to eat healthily, have to take time off work and feel a sense of grief or loss for what they perceive to be a normal pregnancy. "It is therefore vital that women with this condition are given the right information and support and are made aware of the therapeutic and alternative therapies available to help them cope."

‘***http://www.eurekalert.org/pub_releases/2016-06/uot-uot062216.php***

U of T Mississauga professor discovers new origins for farmed rice

Chew on this: rice farming is a far older practice than we knew. In fact, the oldest evidence of domesticated rice has just been found in China, and it's about 9,000 years old.

The discovery, made by a team of archaeologists that includes University of Toronto Mississauga professor Gary Crawford, sheds new light on the origins of rice domestication and on the history of human agricultural practices.

"Today, rice is one of most important grains in the world's economy, yet at one time, it was a wild plant...how did people bring rice into their world? This gives us another clue about how humans became farmers," says Crawford, an anthropological archaeologist who studies the relationships between people and plants in prehistory.

Working with three researchers from the Provincial Institute of Cultural Relics and Archaeology in Zhejiang Province, China, Crawford found the ancient domesticated rice fragments in a probable ditch in the lower Yangtze valley.

They observed that about 30 per cent of the rice plant material - primarily bases, husks and leaf epidermis - were not wild, but showed signs of being purposely cultivated to produce rice plants that were durable and suitable for human consumption.

Crawford says this finding indicates that the domestication of rice has been going on for much longer than originally thought. The rice plant remains also had characteristics of japonica rice, the short grain rice used in sushi that today is cultivated in Japan and Korea.

Crawford says this finding clarifies the lineage of this specific rice crop, and confirms for the first time that it grew in this region of China.

Crawford and his colleagues spent about three years exploring the five-hectare archaeological dig site, called Huxi, which is situated in a flat basin about 100 metres above sea level. Their investigations were supported by other U of T Mississauga participants - anthropology professor David Smith and graduate students Danial Kwan and Nattha Cheunwattana.

They worked primarily in early spring, fall and winter in order to avoid the late-spring wet season and excruciatingly hot summer months.

Digging 1.5 metres below the ground, the team also unearthed artifacts such as sophisticated pottery and stone tools, as well as animal bones, charcoal and other plant seeds.

This study builds on Crawford's previous research into early agriculture in China, in which he has examined the ancient settlements, tools, and plant and animal management efforts that occurred in different regions of the country.

He is interested in better understanding the forces that compelled our human ancestors to transition from hunters and gatherers to farmers.

"The question I ultimately want to answer is, what pushed them to move wholeheartedly into the farming regime? Why did they reduce their emphasis on hunting and expand into crop production?" Crawford says.

"People did what they needed to do to make their lives more manageable and sustainable, and the unintended consequence was farming. With this rice discovery, we're seeing the first stages of that shift."

Funded by a grant from the Social Sciences and Humanities Research Council, Crawford's study is published today in Scientific Reports, an online open-access journal from the publishers of Nature.

http://www.eurekalert.org/pub_releases/2016-06/aqs-soa062216.php

Some older adults live well, despite advancing years and the burdens of chronic diseases

Undefined coping mechanism may play a role in how well older adults are able to live in spite of burdensome illnesses

You might believe that older adults who deal with extensive chronic illnesses or serious diseases would be more likely to be frail and to have a poorer quality of life than healthier older adults.

That may be true for some elders--but not for all. Researchers writing in the Journal of the American Geriatrics Society suggest that an undefined coping mechanism of some sort may play a role in how well older adults are able to live despite having burdensome illnesses.

The researchers examined three groups of participants enrolled in the Cardiovascular Health Study, a large research project that examined adults 65-years-old and older from four cities around the country.

Researchers assigned people to one of three groups, based on the extent of their disease and their level of vigor or frailty:

1. The expected agers (3,528 people) had higher disease but also higher frailty levels. They spent 47 percent of the remainder of their lives able and healthy.

2. The adapters (882 people) had higher disease levels as well as relatively high vigor (being active and mobile) levels. They spent 55 percent of the reminder of their lives able and healthy.

3. The prematurely frail (885 people) had lower disease levels but higher frailty levels. They spent 37 percent of their remaining lives able and healthy.

The researchers said "adapter" older adults who were more vigorous than expected, based on their disease burden, lived longer lives when compared to those who were more frail than expected based on their disease burden.

These "adapters" could have unique characteristics, perhaps some undefined coping mechanism, that should be studied further, suggested the researchers.

This summary is from "Effects of Disease Burden and Functional Adaptation on Morbidity and Mortality." It appears online ahead of print in the June 2016 issue of the Journal of the American Geriatrics Society. The study authors are Jason L. Sanders, MD, PhD; Alice M. Arnold, PhD; Calvin H. Hirsch, MD; Stephen M. Thielke, MD; Dae Kim, MD, ScD; Kenneth J. Mukamal, MD, MPH; Jorge R. Kizer, MD, MSc; Joachim H. Ix, MD, MAS; Robert C. Kaplan, PhD; Stephen B. Kritchevsky, PhD; and Anne B. Newman, MD, MPH.

http://www.eurekalert.org/pub_releases/2016-06/bawh-hbm061716.php

Has breast MRI been performed upside down?

New research from Brigham and Women's Hospital finds changes in patient positioning for MRI from imaging to surgery results in deformation and displacement of the tumor during surgery

BOSTON, MA - Magnetic Resonance Imaging (MRI) has been used as an effective tool for cancer evaluation and has been found to be highly sensitive in detecting breast tumors, but there is no evidence that pre-operative MRI translates into improved outcomes following breast conserving surgery.

Traditionally, patients who are scheduled to undergo breast-conserving lumpectomy for breast cancer undergo a breast MRI prior to surgery to help inform the surgeon about the size, shape, and location of the tumor. These MRIs are performed with the patient lying prone, or face down, while the surgery is performed with the patient lying supine, face up.

A new phase 1 clinical trial from Brigham and Women's Hospital published in Radiology on June 22, 2016, evaluated the differences between pre-operative prone and supine MRI exams in 12 women undergoing lumpectomy for breast cancer. Researchers demonstrated that considerable deformity of the breast and tumor position occurs when patients are imaged in the prone position.

"Accounting for change in size and shape caused by displacement and deformation of the tumor between standard imaging in the prone position and operative supine position, our analysis highlights that supine MRI before surgery may provide surgeons with more detailed and accurate information and could lead to effective tumor removal," stated Eva C. Gombos, MD, radiologist at BWH and lead author of the study.

"Supine MRI, when performed in addition to standard prone breast MRI, may help detect a remnant tumor and ensure clear margins to prevent re-operation. Among women undergoing breast conserving surgery, 15-40 percent need to have a second operation to remove remnant tumor," said senior author Mehra Golshan, MD, distinguished chair in surgical oncology at BWH.

Between April 2012 and December 2014, a total of 15 women were enrolled in the trial in the Advanced Multi-Modality Image Guided Operating Suite (AMIGO) at BWH, an operating room and interventional suite facility with a full array of imaging modalities for use during surgical procedures, designated as the Center for Image Guided Therapeutics by the National Cancer Institute.

All images and information relevant to the procedure are accessible in the operating suite, allowing radiologists and surgeons to continuously view relevant imaging data. Integration of intra-operative MRI for breast lumpectomy was developed by the late Ferenc A. Jolesz, MD, former director of the MRI Division, and the Image-Guided Therapy Program at BWH.

Patients in the study underwent standard diagnostic MR imaging in the usual prone position as an outpatient preceding surgery.

Twelve patients underwent lumpectomy and post-surgical supine MRI during the operation. Half had pre-procedure supine imaging. Researchers measured differences found in size, position, and shape of tumor between prone and supine imaging.

Researchers found that specifications of the tumor, including size and location in the breast, were substantially different depending on the position of the women when she had her MRI. All patients underwent successful removal of their tumor with clear margins for invasive breast cancer.

"If validated in future large studies, intra-operative, and, more importantly, pre-operative supine MRI could be expected to help the surgeon in accurately planning removal of the tumor and reducing the need for re-operation which negatively impacts the patient emotionally, delays post-operative therapy and increases infection rates and cost," stated Gombos.

This study was supported by the National Center for Research Resources and the National Institute of Biomedical Imaging and Bioengineering of the National Institutes of Health through Grant Numbers P41EB015898 and P41RR019703. The National Institute of Health Grant R25 CA089017 by the Breast Cancer Research Foundation grant also provided grant support.

http://www.eurekalert.org/pub_releases/2016-06/isoa-ys062216.php

'Hey! You stole my food!'

Abnormal eating behaviors in frontotemporal dementia

Frontotemporal dementia is associated with a wide variety of abnormal eating behaviors such as hyperphagia, fixations on one kind of food, even ingestion of inanimate objects, making an already difficult situation even worse.

A review by SISSA researchers gathers together the state of the art of what is known in this field, paying particular attention to the brain mechanisms involved. The information may be used for understanding eating disorders in healthy people. The review was published in the magazine Neurocase.

The "Banana lady" described by Andrew Kertesz ("The Banana Lady and Other Stories of Curious Behavior and Speech," 2006) ate only bananas and drank liters and liters of milk every day. She continually asked her husband to make sure that there was always enough milk and bananas in the house.

After her death, brain analysis confirmed her doctors' diagnosis: the woman was suffering from frontotemporal dementia, a common type of dementia second only

to Alzheimer's. Alterations in eating behavior are so frequent in this disease that they are factored into the diagnosis.

A systematic review by SISSA Researcher, Marilena Aiello, in collaboration with Vincenzo Silani (IRCCS Istituto Auxologico Milan) and Raffaella Rumiati, SISSA professor and coordinator of the iNSuLa laboratory (Neuroscience and Society), provides an overview of the research done in this field, creating a comprehensive framework to help establish the state of the art and suggest new lines of research.

"We put together what appeared to be a fragmented image, focusing on types of disorders and hypotheses about the brain mechanisms behind them," says Aiello. "This could be helpful for understanding abnormal eating behavior in healthy people as well."

There are many kinds of disorders described in the literature, ranging from a simple increase in appetite, to uncontrolled overeating, lack of satiety, changes in food preferences as in the extreme example of the so-called "Banana lady," and ingesting objects .

There have been other rather extravagant behaviors related to food observed as well, such as stealing food from other peoples' plates. "These behaviors are problematic, of course, socially, but also with regard to patients' health as they tend to gain weight," says Aiello, "even if individual consequences are different. Some people lose weight because they eat a narrow range of foods in an obsessive way."

From an analysis of the studies in the review, there is a link with certain areas of the brain, including the orbitofrontal cortex and most probably the hypothalamus, an area of the brain that regulates the interaction between the amount of food consumed and energetic homeostasis.

"The origin of food anomalies in frontotemporal dementia is likely due to many factors," says Aiello. "It may involve an alteration of the autonomic nervous system, characterized by an altered assessment of the body's signals, such as hunger, satiety, and appetite. Damage to the hypothalamus can cause a loss of inhibitory signals, causing behaviors such as overeating."

"There are probably sensory and cognitive factors that can complicate the picture, continues Aiello. "In patients who eat objects, for example, there is perhaps a semantic problem of recognizing the object of and its function."

"All of these mechanisms," concludes Aiello, "are interesting for understanding the disease and creating optimal treatments to counteract symptoms. At the same time, they reveal abnormalities that may be present, albeit with varied intensities, in healthy individuals with irregular eating habits."

http://www.eurekalert.org/pub_releases/2016-06/w-ldo062216.php

Low doses of common cancer drug may promote cancer spread

At low doses paclitaxel promotes cancer's spread to the liver

New research indicates that paclitaxel, which is the most commonly used chemotherapy for breast cancer, suppresses tumors when given at a certain dosage, but at low doses, it actually promotes cancer spread to the liver.

The findings suggest that lowering the dose of paclitaxel to reduce toxic side-effects is not a safe strategy.

"Paclitaxel and its analogous compounds are the first line agents widely used in clinical cancer chemotherapy. However, potential risks and reasonable treatment strategies of paclitaxel continue to be widely investigated," wrote the authors of The FEBS Journal study.

http://www.eurekalert.org/pub_releases/2016-06/q-slo062216.php

Study links omega-3s to reduced mortality

Meta analysis shows 9 percent reduced risk associated with omega-3 intake

A recent meta-analysis in Scientific Reports supports a link between EPA and DHA omega-3 intake and a reduced risk of death by any cause. The meta-analysis included 11 studies involving 371,965 participants and 31,185 death events, with a subset of the studies being used for different analyses.

In the analysis of n-3 LCPUFA intake, there was a 9% reduced risk of all-cause death associated with high versus low omega-3 intake. In the dose-response analysis, an increase in EPA/DHA intake of 300 mg/day was associated with a 6% lower risk of all-cause mortality. These findings suggest that both dietary and circulating n-3 LCPUFA are shown to be significantly associated with reduced risk of all-cause mortality.

According to study author Manfred Eggersdorfer, "The meta-analysis of 11 prospective observational studies demonstrates that each 1% increment of omega-3s in total fatty acids in blood may be associated with a 20% decrease in risk of all-cause mortality. This is an important finding for the potential contribution of adequate omega-3 intake to public health."

http://www.eurekalert.org/pub_releases/2016-06/jhu-haw062216.php

How a woman with amnesia defies conventional wisdom about memory

She no longer recognizes a Van Gogh, but can tell you how to prepare a watercolor palette.

She can't recall a single famous composer, but knows the purpose of a viola's bridge. She hasn't flown a plane since 2007, when viral encephalitis destroyed her hippocampus, the part of the brain used to form new memories and retrieve old

ones. And she couldn't describe a single trip she's ever taken. But in detail, she'll list the steps needed to keep a plane from stalling and where to find the rudder controls.

Johns Hopkins University cognitive scientists say the sharp contrasts in this patient's memory profile -- her inability to remember facts about pursuits once vital to her life as an artist, musician and amateur aviator, while clearly remembering facts relevant to performing in these domains -- suggest conventional wisdom about how the brain stores knowledge is incorrect.

Conventional wisdom about memory firmly separates declarative knowledge, or memories about facts, from memories for skills, or "muscle memory." For instance, a severe amnesiac with muscle memory might never forget how to ride a bike, but probably couldn't recall anything about the Tour de France. But because skilled performance, like playing music or flying airplanes, requires much more than mere muscle memory, and because this patient retained it despite losing most other aspects of her declarative memory, researchers conclude this type of skill-related declarative knowledge is different.

"There is such a contrast between her not being able to tell us anything about her former life and not being able to tell us anything about many aspects of art and music that she once knew well, but when we ask her to tell us how to do a watercolor, she is articulate and full of detail," said Barbara Landau, the Dick and Lydia Todd Professor of Cognitive Science at Johns Hopkins. "How can you talk about this knowledge of "how to" as distinct from declarative knowledge? It is declarative knowledge."

The findings, now online, are due to appear in an upcoming issue of the journal Cognitive Neuropsychology.

Before her illness, Lonni Sue Johnson, 64, was an accomplished artist whose portfolio included six New Yorker magazine covers. She was also an amateur violist who played in orchestras and chamber groups and a licensed single-engine airplane pilot who flew more than 400 flights and owned two planes. Her illness left her with severe brain damage and catastrophic memory impairment, including severe losses of memory about her previous life and severely restricted ability to learn new facts.

She has very little memory of her past -- not even of her wedding day. She forgets having done something immediately after doing it. She also has very little memory for general world knowledge, including facts about the fields in which she once excelled.

To determine whether Johnson's "skill-related" memory was preserved despite extensive losses in memory for general world knowledge, the team tested her on her memory for facts related to performing four of her former top skills -- art, music, flying and driving. They gave the same tests to people of similar age and experience in those areas, as well as to people with no experience in them.

The oral tests, of about 80 questions each, covered information about the techniques, equipment and terminology involved in performing the various skills. They included queries such as "How might one remove excess paint when painting with watercolor?" and "How should one touch the strings of an instrument to produce a harmonic?"

In art and driving, Johnson scored nearly as high as experts taking the test. In music and aviation, she did not perform as well, but knew considerably more than the novices.

"Although Johnson had not created watercolors, had not flown a plane, and had not driven since her illness, she could still describe how one would go about carrying out these activities," said Johns Hopkins cognitive scientist Michael McCloskey. "These findings suggest that skill-related knowledge can be spared even with dramatic losses in other kinds of knowledge."

The team also included first author Emma Gregory, a former Johns Hopkins post-doctoral fellow, and research assistant Zoe Ovans, also of Johns Hopkins.

This research was supported by the Brain Science Institute at Johns Hopkins.

http://www.eurekalert.org/pub_releases/2016-06/cumc-sfc062116.php

Study finds contagious cancers are spreading among several species of shellfish

New research suggests that direct transmission of cancer among marine animals may be much more common than once thought.

VIDEO: ***https://youtu.be/Ir5H-yZONq8***

Direct transmission of cancer among some marine animals may be more common than once thought, suggests a new study published in Nature by researchers at Columbia University Medical Center (CUMC).

The study, led by Stephen Goff, PhD, the Higgins Professor of Biochemistry in the Department of Biochemistry & Molecular Biophysics and the Department of Microbiology & Immunology at CUMC in collaboration with researchers from Canada and Spain, revealed that in several species of bivalves, including mussels, cockles, and clams, cancer cells from contagious cells that spread from animal to animal through the sea water. The cancer, known as disseminated neoplasia, is a leukemia-like disease that affects bivalves in many parts of the world.

Direct transmission of cancer cells is quite rare. Until recently, the phenomenon had only been observed in two species of mammals.

Last year, Dr. Goff's team found a third example in the soft shell clam (Mya arenaria) after initially suspecting that the culprit behind the cancer cluster was a virus.

The team then wondered if cancers in other mollusks are also caused by contagious cells. To find out, Dr. Goff's team examined the DNA of cancers and normal tissue from mussels (Mytilus trossulus), cockles (Cerastoderma edule), and golden carpet shell clams (Polititapes aureus) collected from the coasts of Canada and Spain.

In each species, the researchers discovered that the cancers were caused by independent clones of cancer cells that were genetically distinct from their hosts. They also found that in one species, the carpet shell clam, the infectious cancer cells came from a related but distinct species. The researchers concluded that this cancer was due to a case of cross-species transmission.

"Now that we have observed the spread of cancer among several marine species, our future research will investigate the mutations that are responsible for these cancer cell transmissions," said Dr. Goff.

The study is titled, Widespread transmission of independent cancer lineages within multiple bivalve species. Additional authors included Michael Metzger (Columbia University Medical Center and Howard Hughes Medical Institute, New York, NY); Antonio Villalba (Centro de Investigacións Mariñas, Vilanova de Arousa, and University of Alcalá, Alcalá, Spain); Maria J. Carballal, and David Iglesias (Centro de Investigacións Mariñas); James Sherry and Carol Reinisch (Environment Canada, Burlington, Ontario, Canada); Annette Muttray (University of British Columbia and SLR Consulting Canada, Vancouver, Canada); and Susan Baldwin (University of British Columbia).

Support for the study was provided by the Howard Hughes Medical and Training Grant T32 CA009503. Additional support was provided by the Consellería do Mar da Xunta de Galicia, project PGIDIT-CIMA 13/03.The researchers declare no conflicts of interest.

http://bit.ly/28TRIHt

Why you should worry about intelligent machines

Artificial intelligence itself isn't a problem – the threat lies in what humans might do with it

THEY started off by wounding our pride. Will AI end up taking our jobs – or even our lives?

Twenty years ago, IBM's Deep Blue beat Garry Kasparov at chess – then seen as the gold standard of human intellect. Now a new wave of AI seems poised to take over a wide range of human tasks, potentially putting huge numbers of people out of work. And an unlikely alliance of philosophers, technologists and movie-makers has stoked fears that the next generation of AI might snuff out humanity.

A reality check is needed. We are nowhere near the creation of a machine that replicates the full suite of a human's intellectual capabilities. And the threat of extinction by superintelligences, if and when they arrive, is only one of a number of esoteric possibilities (see "Forget killer robots: This is the future of supersmart machines").

So where does paranoia about AI come from? In part, it's the challenge smart machines present to long-standing ideas about human exceptionalism, which survived the Copernican and Darwinian revolutions but may be fatally undermined by intelligent – or even conscious – machines. It's also a type of techno-pessimism: we can foresee the potential downsides, but the upsides aren't yet clear.

That doesn't mean the boom in AI gives us nothing to worry about. As ever, it isn't the technology itself that should concern us, but how humans design and use tools based on it. "AI should be used to upskill workers rather than paring their jobs back to tedious piecework"

On the issue of human exceptionalism, there's not a lot that can be done. Even cherished qualities like creativity and invention may well be outsourced to AI in the coming years. But we shouldn't feel threatened by this: we should feel exhilarated at the new things we can do with their help, just as the digital tools we use today have enhanced and diversified the ways in which we communicate and create.

When it comes to jobs, the AI threat is probably overstated. On closer scrutiny, many seemingly straightforward jobs include cognitively taxing elements that AI cannot master – yet (see "Find your meaning at work: 6 things a salary can't buy"). The "gig economy", pioneered by firms such as Uber, adds flexibility to the labour force and convenience to their customers via algorithmic management – but at a cost to workers' rights and conditions. AI could accelerate that trend. That matters: our work is integral to our identities, and preserving the dignity of labour should be central to our society (see "Don't give up the day job: Why going to work is good for you"). We should strive to ensure that AI is used to upskill workers rather than paring their jobs down to tedious piecework: dehumanising workers is a poor use of the technology.

Tackling that is a social and political issue, rather than a technological one. AI may force changes on our economic system – witness the discussions over the introduction of a basic universal income for all (see "What happens if we pay everyone just to live?"). But change should be people-centric, not led by AI-driven efficiency that enriches a few to the detriment of the many.

As for super-smart AI wiping us out, relax. For the moment we should worry more about semi-smart machines given too much power. Autonomous weapons may be in legal limbo, but that hasn't stopped their development. Drone warfare provides a taste of what's to come: how can machines that can't tell civilians from combatants do the "right" thing without human control?

In this, as elsewhere, the answers lie with us. AI can't strip us of our jobs, our dignity or our human rights. Only other humans can do that.

http://bit.ly/28Y1Cbx

WHO Says Saudi Misdiagnosis Caused MERS Outbreak

At least 49 patients and medical staff were exposed

(Reporting by Tom Miles; Editing by Janet Lawrence)

The wrong diagnosis of a woman suffering from the MERS coronavirus led to more than 49 other patients and medical staff being exposed to the disease in a Saudi hospital, the World Health Organization said in a statement on Tuesday.

The unnamed 49-year-old from Buridah city developed symptoms on June 9 and was admitted to hospital on June 10 where she was in a critical condition in an intensive care unit, the WHO said.

On June 12, she tested positive for Middle East Respiratory Syndrome (MERS), a potentially fatal coronavirus from the same family as the one that caused China's deadly 2003 outbreak of Severe Acute Respiratory Syndrome (SARS). The WHO said the woman had initially gone to hospital for a condition unrelated to MERS.

"She was then admitted to the vascular surgery ward – MERS-CoV infection was not considered. She was not isolated and was managed in a multi-bed room. During this time, more than 49 HCWs (healthcare workers) and patients were exposed," the WHO said. A rapid response team immediately tried to trace the people with whom she had had contact at her home or in the hospital in Riyadh, and 20 of them tested positive, although 18 of the 20 had no symptoms.

The Saudi Health Ministry had no immediate comment on the WHO statement.

MERS is thought to be linked to camels and consumption of camel milk, but most of the known human-to-human transmission has occurred in health care settings, and the WHO has said hospitals and medical workers should take stringent precautions as a standard measure to stop the disease spreading.

WHO Director-General Margaret Chan has previously criticized Saudi Arabia for allowing MERS to spread in its hospitals, which showed infection control standards were not being adhered to. But she said last October new Saudi Health Minister Khaled al-Falih, was "much more forthcoming" than his predecessor.

Since September 2012, WHO has been notified of 1,761 laboratory-confirmed cases, including at least 629 related deaths.

http://www.medscape.com/viewarticle/864887

The 'Good Old Days' of Paper Charts

The Hidden Value of a Patient's Social History

Greg A. Hood, MD|June 22, 2016

Cynics and neo-techno luddites will forever lament the loss of the elegance and availability of the written word, particularly when it comes to the paper medical chart. There are some things to be said for this position. But, to be fair, there have been opportunities that were missed or not taken advantage of in both the eras of the written chart and the electronic health record (EHR).

In particular, the social history section has long been misunderstood and underutilized. No, I'm not going to enter into the old axiomatic debate of whether substance use belongs in the medical history or social history. Instead, I'm speaking of taking full advantage of the "social" aspect of the social history.

Throughout my more than 20-year career, I've used the social history to keep track of notes that help personalize my relationship with patients and provide them with good service.

I'm talking about such things as weddings, births, anniversaries, recent vacations, and anything else that might be important or represent a milestone in a patient's life.

Thankfully, I've been able to maintain my methods with our current office system. However, between the "usability"/ease of interface of some EHR systems, and the ever-increasing time pressures that the system and its documentation requirements exert, the social history is becoming more of an anticlimactic social history. But I digress.

Providing good service should be a priority for all physicians. Patients, of course, hope to receive good service. Old and well-cited studies have shown that patients can't always accurately judge the quality of the care that they receive. However, they can recognize personalized service, be it from a hotel, a restaurant, or a physician's office. They also often equate personalization of care with quality of care.

What You Gain by Taking a Good Social History

It has become de rigueur to note the usual details in the social history. However, because this is the only appropriate area of the chart to log other nonmedical details, recording other information can quickly help each provider, or even nonclinical staff, to provide more personalized care.

Being able to easily retrieve such details as the name of a beloved pet, or asking about grandkids who are in college, can garner quick and ready positive attention.

This approach can also be of benefit in those cases of "same name" conflicts. For instance, remembering by a glance which John Smith has a particular interest, or even a hobby that he shares with you, can be very helpful.

As physicians, we take care of thousands of patients. Recording these details can help you keep things straight, even when you're tired, behind schedule, or preoccupied trying to figure out a complex medical problem.

Remembering to throw in a question about a family member or remembering the destination of an anniversary trip are the sorts of details that all physicians hope to

recall when engaging in personalized care with their patients. Systematizing this approach is a provider's only hope in today's fast-paced medical climate.

The social history is the best place to store them, because most EHRs allow for free-text entry in this area, if no other. Unfortunately, these machines often make it difficult to enter such information, bringing our profession one step closer to treating patients like they, too, are machines.

The Reason We Entered Medicine in the First Place

Taking the time and making the effort to really get to know patients isn't merely a series of one-way interactions. Spending 30 seconds broaching personal topics can preserve the one type of enjoyment for which we all entered the profession in the first place.

If these interactions don't come easily to you, I suggest you read Olivia Fox Cabane's The Charisma Myth: How Anyone Can Master the Art and Science of Personal Magnetism (Portfolio/Penguin, 2012). This book can help you examine your own capabilities in interacting with patients and help you address any insecurities you might have.

The freedom to make personal connections with our patients is the reason why physicians sought out healthcare rather than finance, IT, or some other high-paying (or higher-paying) profession.

This degree of personalization also helps to balance the social interactions between physicians and patients, who have come to share and discuss elements of their lives that perhaps no one else may even know about. Chatting with them about nonmedical topics—even if it's for just a minute—helps patients understand that they're empathized with as a whole person, not just as a list of problems or an organ system.

Although I make a concerted effort to collect the details of my patients' lives, I've never been accused of doing this on purpose. Even if you do get "caught" referring to your notes, what patient could object to you and your staff making the time and effort to take such an interest?

In some cases, especially when you're scrambling to stay on schedule, attending to such discussion points also helps patients feel you spent more time with them than you actually did.

In short, the physician who includes a brief social interaction during the office visit is judged by patients to have taken more time with them and to have delivered better care. Properly motivated, caring, personal interactions like the ones I've described—in medicine and in our lives in general—facilitate the lubrication upon which a civilized society functions.

http://www.eurekalert.org/pub_releases/2016-06/uol-nfc062216.php

New findings challenge current view on origins of Parkinson's disease

MRC researchers at University of Leicester investigate 'mutant flies'

The neurodegeneration that occurs in Parkinson's disease is a result of stress on the endoplasmic reticulum in the cell rather than failure of the mitochondria as previously thought, according to a study in fruit flies. It was found that the death of neurons associated with the disease was prevented when chemicals that block the effects of endoplasmic reticulum stress were used.

Some inherited forms of early-onset Parkinson's disease have typically been blamed on poorly functioning mitochondria, the powerhouses of cells. Without reliable sources of energy, neurons wither and die. This may not be the complete picture of what is happening within cells affected by Parkinson's. Researchers from the MRC Toxicology Unit at the University of Leicester used a common fruit fly to investigate this further; fruit flies were used because they provide a good genetic model for humans.

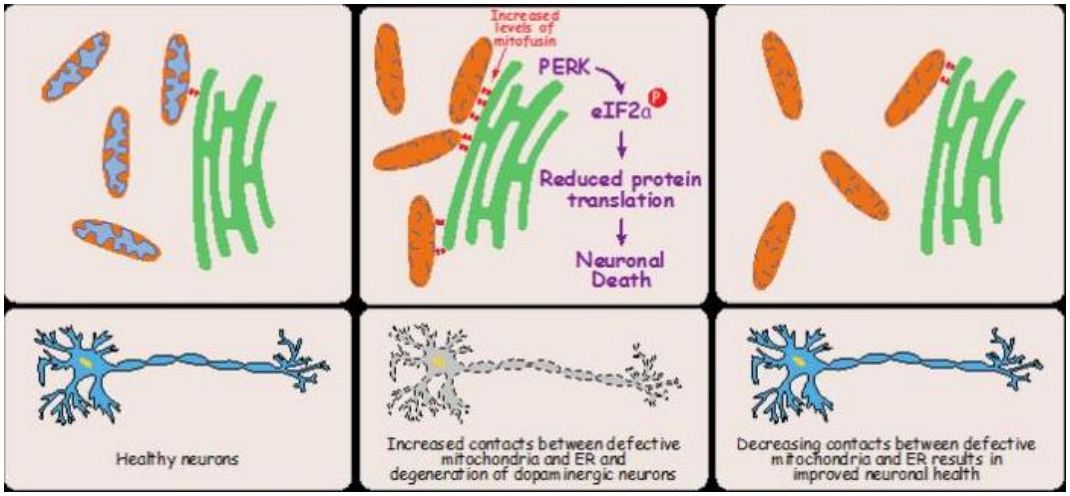


Image shows a summary of our main findings. Mitochondria in orange and ER in green. **University of Leicester**

Studies on human subjects are of limited use for elucidating the signaling pathways and cellular processes underlying the neurodegenerative process. This is because both ethical and technical constraints limit the extent to which genetic analysis can be performed in humans.

Flies are a well-established model animal to understand the molecular mechanisms of human diseases. This is because about 75% of human disease-causing genes are found in the fly in a similar form. Also, they are easy to work

with, breed quickly and many tools are available to manipulate any genes in the fly. In flies, potential therapeutic drugs can be mixed with food and readily tested.

It was found that the bulk of the damage to neurons with damaged mitochondria stems from a related but different source - the neighbouring maze-like endoplasmic reticulum (ER).

The ER has the important job of folding proteins so that they can do the vast majority of work within cells. Misfolded proteins are recognized by the cell as being dangerous. Cells halt protein production if there are too many of these harmful proteins present. While this system is protective, it also stalls the manufacture of vital proteins, and this eventually results in the death of neurons.

To find out if ER stress might be at play in Parkinson's, a team led by Dr Miguel Martins analyzed fruit flies with mutant forms of the pink1 or parkin genes. Mutant forms of pink1 and parkin are already known to starve neurons from energy by preventing the disposal of defective mitochondria. These genes are also mutated in humans and result in hereditary versions of the disease. Much like Parkinson's patients, flies with either mutation move more slowly and have weakened muscles. The insects struggle to fly and they lose dopaminergic neurons in their brains - a classic feature of Parkinson's.

Compared to normal flies, Miguel's team found that the mutants experienced large amounts of ER stress. The mutant flies did not manufacture proteins as quickly as the non-mutants. They also had elevated levels of the protein-folding molecule BiP, a telltale sign of stress.

One function of pink1 and parkin genes is to help degrade mitofusin - a protein that tethers the endoplasmic reticulum to mitochondria. Mutant flies have an abundance of this protein. It was found that the mutants had more of their mitochondria attached to the ER than normal flies. For this reason, the researchers suggest that ER stress is related to extra tethering of mitochondria, thereby preventing the removal of defective versions of the organelle.

Mutant flies, which have more of these tethers, have fewer dopaminergic neurons, which can have an adverse effect on the brain. By reducing the number of these tethers it is possible to prevent the loss of the neurons. When the researchers experimentally lowered the amount of mitofusin in the mutants, the number of tethers fell and the neuron number increased again (see figure). The flies' muscles also remained healthy despite the mitochondria themselves still being defective.

These results suggest that the neurodegeneration seen in Parkinson's is a result of ER stress rather than a general failure of the mitochondria. The scientists were able to prevent neurodegeneration in mutant flies not only by reducing mitofusin, but also with chemicals that block the effects of ER stress.

Dr Miguel Martins said: "This research challenges the current held belief the Parkinson's disease is a result of malfunctioning mitochondria. By identifying and preventing ER stress in a model of the disease it was possible for us to prevent neurodegeneration. Lab experiments, like this, allow us to see what effect ER stress has on Parkinson's disease. While the finding so far only applies to fruit flies, we believe further research could find that a similar intervention in people might help treat certain forms of Parkinson's."

The research has been published in the journal Cell Death and Disease and a video explaining the main findings can be found here
https://www.youtube.com/watch?v=13oA5eiH-8s

http://bit.ly/28WnBMT

New life form discovered in saliva is linked to human disease

Attack of the microbes

By Andy Coghlan

Parasitic bacteria that are entirely dependent on the other bacteria they infect have been discovered for the first time, in human spit. The tiny cells have gone undetected for decades, but appear to be linked to gum disease, cystic fibrosis and antimicrobial resistance.

We only know of one other strain of bacteria that can infect other bacteria, but this type, called Bdellovibrio, is a free-living cell that hunts down its prey. The newly discovered organism has very few genes and is dependent entirely on its host.

The parasite, which appears to make its host more harmful to humans, evaded our detection until now because it is difficult to grow and study in the laboratory.

“They’re ultra-small bacteria, and live on the surface of other bacteria,” Jeff McLean of the University of Washington School of Dentistry in Seattle told the annual meeting of the American Society for Microbiology in Boston, Massachusetts, last week.

Unlike any known species

McLean and his colleagues discovered the organisms by searching for bacterial strains in human saliva samples.

Analysing the DNA of all the species they managed to grow from these samples, they came across a mystery fragment of genetic material. This piece of RNA had been discovered by other researchers before, but no one could tell what organism it came from.

McLean’s team has now shown that this RNA belongs to a bacterium that lives on another species, Actinomyces odontolyticus. When the researchers viewed this species under the microscope, they found that the cells were covered with much smaller bacteria – the first species ever discovered to parasitise another bacterium.

At first, Actinomyces is able to tolerate the parasites, which attach themselves to its outer membrane, drawing nutrients out of their host.

"Later, they start attacking and killing the host," said McLean. Towards the end of the infection process, holes seem to form in the membrane of the Actinomyces cell and its contents gush out.

"We're trying to decipher what's going on," he said.

The parasitic bacterium is unlike any previously known species. It has just 700 genes and is the first bacterial strain identified that cannot make its own amino acids – the building blocks for the proteins essential to life – but depends instead on a supply from its host. By comparison, A. odontolyticus has 2200 genes.

Tip of the iceberg

This discovery explains why the species has never been seen before: it can only be grown in the laboratory alongside a host. McLean suspects A. odontolyticus is not this parasite's only host, and that many other types of tiny parasitic bacteria may exist.

"This microbe is clearly the tip of the iceberg," suggests Roland Hatzenpichler of the California Institute of Technology in Pasadena.

"It's incredibly exciting to see such a major advancement in the study of major lineages of life that until now have been impossible to cultivate," says Brian Hedlund of the University of Nevada, Las Vegas. "Gene data from other as-yet uncultivated organisms suggests that host-parasite relationships between microbes are common in nature, so this type of study is a great template for others to follow."

Disease and antibiotic resistance

We might find that these species have an important role in human diseases. McClean says he has found high concentrations of the new bacterium's DNA in people who have gum disease or cystic fibrosis.

Actinomyces bacteria are known to contribute to gum disease, but are usually kept under control by white blood cells called macrophages, which engulf and destroy them. McLean says he has evidence that when these bacteria are infected with the parasite, they can evade these macrophages and make gum disease worse.

In previous work, the team had identified a type of bacterium that infects some members of the archaea – a different type of single-celled life that is genetically distinct from bacteria, but similar in its lack of a true cell nucleus and other complex cell machinery.

The two parasitic bacteria also both somehow make their host cells become resistant to the antibiotic streptomycin – another finding that may prove important in the midst of our present antimicrobial-resistance crisis.

http://www.eurekalert.org/pub_releases/2016-06/aaft-wfd062016.php

Why fathers don't pass on mitochondria to offspring

New study reveals how and why mitochondria are only passed on through a mother's egg - and not the father's sperm

Offering insights into a long-standing and mysterious bias in biology, a new study reveals how and why mitochondria are only passed on through a mother's egg - and not the father's sperm.

What's more, experiments from the study show that when paternal mitochondria persist for longer than they should during development, the embryo is at greater risk of lethality.

Harbored inside the cells of nearly all multicellular animals, plants and fungi are mitochondria, organelles that play an important role in generating the energy that cells need to survive.

Shortly after a sperm penetrates an egg during fertilization, the sperm's mitochondria are degraded while the egg's mitochondria persist.

To gain more insights into this highly specific degradation pattern, Qinghua Zhou et al. used electron microscopy and tomography to study sperm mitochondria (or paternal mitochondria) in Caenorhabditis elegans, a type of roundworm, during early stages of development.

Intriguingly, the paternal mitochondria were found to partially self-destruct before the mitochondria were surrounded by autophagosomes, which target components within a cell and facilitate their degradation.

This suggests that another mechanism, something within the paternal mitochondrion itself, initiates the degradation process.

RNA analysis of paternal mitochondria during early stages of embryonic development hinted that it is the cps-6 gene that facilitates this process, which the team confirmed by studying sperm lacking cps-6; without it, paternal mitochondria remained significantly later into the development stage.

Further investigation suggests that the enzyme that cps-6 encodes first breaks down the interior membrane of the paternal mitochondria before moving to the space within the inner membrane to breakdown mitochondrial DNA.

When the researchers engineered paternal mitochondria to breakdown during later stages of development, this increased the chances that the embryo would not survive, suggesting that the transmission of paternal mitochondria is an evolutionary disadvantage.

Collectively, results from this study suggest that cps-6 plays a key role in initiating the self-destruction of paternal sperm, which likely benefits the embryo.

http://www.eurekalert.org/pub_releases/2016-06/uota-ff3062216.php

Fix for 3-billion-year-old genetic error could dramatically improve genetic sequencing

Will increase precision in genetic research and could dramatically improve medicine based on a person's genetic makeup

For 3 billion years, one of the major carriers of information needed for life, RNA, has had a glitch that creates errors when making copies of genetic information. Researchers at The University of Texas at Austin have developed a fix that allows RNA to accurately proofread for the first time. The new discovery, published June 23 in the journal Science, will increase precision in genetic research and could dramatically improve medicine based on a person's genetic makeup.

Certain viruses called retroviruses can cause RNA to make copies of DNA, a process called reverse transcription. This process is notoriously prone to errors because an evolutionary ancestor of all viruses never had the ability to accurately copy genetic material.

The new innovation engineered at UT Austin is an enzyme that performs reverse transcription but can also "proofread," or check its work while copying genetic code. The enzyme allows, for the first time, for large amounts of RNA information to be copied with near perfect accuracy.

"We created a new group of enzymes that can read the genetic information inside living cells with unprecedented accuracy," says Jared Ellefson, a postdoctoral fellow in UT Austin's Center for Systems and Synthetic Biology. "Overlooked by evolution, our enzyme can correct errors while copying RNA."

Reverse transcription is mainly associated with retroviruses such as HIV. In nature, these viruses' inability to copy DNA accurately may have helped create variety in species over time, contributing to the complexity of life as we know it.

Since discovering reverse transcription, scientists have used it to better understand genetic information related to inheritable diseases and other aspects of human health. Still, the error-prone nature of existing RNA sequencing is a problem for scientists.

"With proofreading, our new enzyme increases precision and fidelity of RNA sequencing," says Ellefson. "Without the ability to faithfully read RNA, we cannot accurately determine the inner workings of cells. These errors can lead to misleading data in the research lab and potential misdiagnosis in the clinical lab."

Ellefson and the team of researchers engineered the new enzyme using directed evolution to train a high-fidelity (proofreading) DNA polymerase to use RNA templates. The new enzyme, called RTX, retains the highly accurate and efficient proofreading function, while copying RNA. Accuracy is improved at least threefold, and it may be up to 10 times as accurate. This new enzyme could enhance the methods used to read RNA from cells.

"As we move towards an age of personalized medicine where everyone's transcripts will be read out almost as easily as taking a pulse, the accuracy of the sequence information will become increasingly important," said Andy Ellington, a professor of molecular biosciences. "The significance of this is that we can now also copy large amounts of RNA information found in modern genomes, in the form of the RNA transcripts that encode almost every aspect of our physiology. This means that diagnoses made based on genomic information are far more likely to be accurate. "

In addition to Ellefson and Ellington, authors include Jimmy Gollihar, Raghav Shroff, Haridha Shivram and Vishwanath Iyer. All are affiliated with the Department of Molecular Biosciences at The University of Texas at Austin.

This research was supported by grants from the Defense Advanced Research Projects Agency, National Security Science and Engineering Faculty Fellows, NASA and the Welch Foundation. A provisional patent was filed on the new sequence of the enzyme.

http://www.eurekalert.org/pub_releases/2016-06/uoc--hbh062216.php

Human brain houses diverse populations of neurons, new research shows

A team of researchers has developed the first scalable method to identify different subtypes of neurons in the human brain.

The research lays the groundwork for "mapping" the gene activity in the human brain and could help provide a better understanding of brain functions and disorders, including Alzheimer's, Parkinson's, schizophrenia and depression.

By isolating and analyzing the nuclei of individual human brain cells, researchers identified 16 neuronal subtypes in the cerebral cortex -- the brain's outer layer of neural tissue responsible for cognitive functions including memory, attention and decision making.

The team, led by researchers at the University of California San Diego, The Scripps Research Institute (TSRI) and Illumina, published their findings in the June 24 online issue of the journal Science.

"We're providing a unified framework to look at and compare individual neurons, which can help us find out how many unique types of neurons exist," said Kun Zhang, bioengineering professor at the University of California, San Diego and a corresponding author of the study.

Researchers can use these different neuronal subtypes to build what Zhang calls a "reference map" of the human brain -- a foundation to understand the differences between a healthy brain and a diseased brain.

"In the future, patients with brain disorders or abnormalities could be diagnosed and treated based on how they differ from the reference map. This is analogous to what's being done with the reference human genome map," Zhang said.

The new study reflects a growing understanding that individual brain cells are unique: they express different types of genes and perform different functions. To better understand this diversity, researchers analyzed more than 3,200 single human neurons in six Brodmann areas, which are regions of the cerebral cortex classified by their functions and arrangements of neurons.

Through an interdisciplinary collaborative effort, the team developed a new method to isolate and sequence individual cell nuclei. TSRI researchers led by neuroscience professor Jerold Chun obtained the samples from a post mortem brain and focused on isolating the neuronal nuclei.

Zhang's lab worked with Fluidigm, a manufacturer of microfluidic chips for single-cell studies, to develop a protocol to identify and quantify RNA molecules in individual neuronal nuclei. Scientists at San Diego-based Illumina sequenced the resulting RNA libraries. Researchers led by biochemistry professor Wei Wang at UC San Diego developed algorithms to cluster and identify 16 neuronal subtypes from the sequenced datasets.

Researchers deciphered what types of genes were "turned on" within each nucleus and revealed that various combinations of the 16 subtypes tended to cluster in cortical layers and Brodmann areas, helping explain why these regions look and function differently.

Neurons exhibited many differences in their transcriptomic profiles -- the patterns of genes that are being actively expressed by these cells -- revealing single neurons with shared, as well as unique, characteristics that likely lead to difference in cellular function.

"We're finding new ways to understand the basic building blocks of the brain," said Blue Lake, a postdoctoral researcher in Zhang's lab and a co-first author of the study. "Our study opens the door to look at global gene expression patterns and how that defines cell types within a normal tissue, which can also be used to see what's abnormal in terms of disease or disorders."

In future studies, researchers aim to analyze neurons in other Brodmann areas of the brain and investigate what subtypes exist in other brain regions. They also plan to study neurons from multiple post mortem human brains (this study only involved one) to investigate neuronal diversity among individuals.

The interdisciplinary research is part of a larger effort under the NIH Single Cell Analysis Program-Transcriptome (SCAP-T) Project -- funded by the NIH Common Fund Program -- to develop new single cell analysis technologies.

Full paper: "Neuronal subtypes and diversity revealed by single-nucleus RNA sequencing of the human brain." Authors of the study are Blue B. Lake, Rizi Ai,* Rui Liu, Andre Wildberg, Derek Gao, Ho-Lim Fung, Song Chen, Wei Wang and Kun Zhang of UC San Diego; Gwendolyn E. Kaeser,* Yun C. Yung, Julian Wong, Allison Chen, Xiaoyan Sheng and Jerold Chun of The Scripps Research Institute; and Neeraj S. Salathia,* Raakhee Vijayaraghavan, Fiona Kaper, Richard Shen, Mostafa Ronaghi and Jian-Bing Fan of Illumina. The work was supported by the NIH Common Fund Single Cell Analysis Program (1U01MH098977).*

http://www.eurekalert.org/pub_releases/2016-06/icl-dve062216.php

Dengue virus exposure may amplify Zika infection

Previous exposure to the dengue virus may increase the potency of Zika infection, according to research from Imperial College London

Previous exposure to the dengue virus may increase the potency of Zika infection, according to research from Imperial College London. The early-stage laboratory findings, published in the journal Nature Immunology, suggests the recent explosive outbreak of Zika may have been driven in part by previous exposure to the dengue virus. The study, which included scientists from Institut Pasteur in Paris and Mahidol University in Bangkok, suggests the Zika virus uses the body's own defences as a 'Trojan horse', allowing it to enter a human cell undetected. Once inside the cell, it replicates rapidly.

Professor Gavin Screaton, senior author of the research and Dean of the Faculty of Medicine at Imperial, said: "Although this work is at a very early stage, it suggests previous exposure to dengue virus may enhance Zika infection. This may be why the current outbreak has been so severe, and why it has been in areas where dengue is prevalent. We now need further studies to confirm these findings, and to progress towards a vaccine."

A second study by the same team, published in Nature, suggests an antibody that works against the dengue virus may also neutralise Zika - providing a potential target for a vaccine.

Dengue fever has risen dramatically over recent decades and the virus is thought to cause around 390 million infections each year - with 40 per cent of the world's population living in areas of risk.

The dengue virus is similar to the Zika virus - they belong to the same viral family, called the Flaviviridae, and both are transmitted by the Aedes mosquito.

In the new Nature Immunology paper, supported by the Wellcome Trust and the Medical Research Council, the researchers used antibodies that recognise the dengue virus collected from individuals who had been infected with dengue. The team, who were also supported by the National Institute for Health Research Imperial Biomedical Research Centre, added them to human cell cultures, together with the Zika virus.

Their results suggest dengue antibodies can recognise and bind to Zika, due to the similarities between the viruses. Crucially, they also suggest that pre-existing dengue antibodies can amplify a Zika infection through a phenomenon called antibody-dependent enhancement (ADE).

This has been previously identified in dengue fever, and is thought to be why a second infection with dengue is often more serious than the first.

When dengue first infects the body, the immune system makes antibodies against the virus. Antibodies are large proteins that latch onto invading bacteria or viruses, neutralising them and enabling the immune system to destroy the pathogens. The antibodies are then primed to recognise the same invaders should another attack occur.

However, there are four different types of dengue virus. If someone is infected a second time by a different strain, the antibodies from the first attack can only partially bind to the virus, and are unable to prevent infection.

The antibody, with the virus loosely attached, then shuttles into an immune cell. This immune cell would normally then kill the virus, but because the virus is not properly attached, it breaks free once it gains entry to the human immune cell. Here it hijacks the immune cell's machinery to replicate more viral particles, enhancing the infection.

The new study suggests the same phenomenon occurs when a person who has previously been exposed to dengue encounters Zika. The existing dengue antibodies latch onto Zika, due to similarity between the viruses. However the antibodies are unable to latch onto Zika securely, and so the antibody simply facilitates entry of Zika into the human immune cells, where it replicates.

"We now need to investigate whether the phenomenon of ADE may aid transfer of Zika across the placenta," explained Dr Juthathip Mongkolsapaya, co-author from the Department of Medicine at Imperial.

She added that the team also found that a type of antibody may help protect against the phenomenon of ADE, and prevent the virus from hijacking the immune cells. Previous work from the team has shown the immune system generates different types of antibodies to dengue that bind to various areas of the virus. In the current study, the team found a group of antibodies that bind to a certain site on the dengue virus - called EDE1 antibodies - were able to prevent the Zika virus from entering the immune cell.

In a second study, published in Nature and co-authored by Professor Felix Rey from the Institut Pasteur and Professor Screaton from Imperial, the team confirmed that EDE1 antibodies bind efficiently to the Zika virus and potently neutralise infection. The team are now working hard to use these findings to develop new vaccines to dengue and Zika.

Dr Jeremy Farrar, Director of the Wellcome Trust, said: "Zika and dengue come from the same family of viruses and we know they share many similarities in their genetic make-up, transmission pattern and in the immune response they trigger. These new studies suggest that prior infection with dengue doesn't offer any protection against Zika, and may in fact predispose people to a more severe infection. We can't say yet whether this interaction is playing a role in the current outbreak, but if confirmed it's likely to have important implications for the control and global spread of Zika, and for the development of any vaccine for the virus. There are still more questions than answers about Zika and this group of viruses including dengue. We know that Zika has been present in Southeast Asia and Africa for many years and yet has not taken off there as it has in South America. This is what the international research effort needs to work out, and quickly."

The work in both papers was supported by The Wellcome Trust, the Medical Research Council, the National Institute for Health Research Imperial Biomedical Research Centre and the European Commission Seventh Framework Programme.

http://www.eurekalert.org/pub_releases/2016-06/ip-ata062316.php

Antibodies that are effective against both dengue and Zika viruses

Identification of antibodies efficiently neutralizing both dengue virus and Zika virus

Scientists from the Institut Pasteur and the CNRS [1], in collaboration with Imperial College London and the University of Vienna, Austria, have identified antibodies that can efficiently neutralize both the dengue virus and the Zika virus. The description of the binding site for these antibodies on the viral envelope, identical for both viruses, could lead to the development of a universal vaccine that offers simultaneous protection against dengue and Zika virus disease. These results were published in the journal Nature on June 23, 2016.

The dengue virus and the Zika virus share several characteristics. They both belong to the Flavivirus genus, they are both RNA viruses mainly transmitted by mosquitoes, and they have similar envelope proteins. Scientists from the Institut Pasteur, the CNRS and Imperial College London, who had previously identified antibodies capable of neutralizing the four types of dengue virus in an earlier study, decided to turn their attention to the Zika virus. "We wanted to see whether the antibodies isolated for dengue could be used to neutralize other flaviviruses, and Zika seemed like the best candidate," explained Félix Rey, Head of the Structural Virology Laboratory at the Institut Pasteur.

In this latest study, the scientists selected two antibodies capable of blocking the proliferation of the dengue virus. They began by isolating these antibodies in dengue patients, then presented them to the Zika virus. One of the antibodies proved to be highly efficient in neutralizing the Zika virus - even more so than for

dengue - preventing it from infecting the cells it was cultured with. "We never expected to discover that the dengue virus and the Zika virus are so close that some antibodies produced against the dengue virus could also neutralize the Zika virus so potently" stressed Félix Rey.

The scientists then used crystallography to identify the antibody binding site on the Zika virus, and more specifically on the proteins in its viral envelope. Crystals containing the "antibody-envelope protein" complex were produced using the Institut Pasteur's Crystallography Platform. The scientists then used powerful X-rays produced by the synchrotron facilities in Saclay and Grenoble to create a 3D reconstruction of the precise location where the antibody binds to the envelope protein.

This revealed that the antibody binding site for the Zika virus is the same as for the dengue virus, raising the possibility of producing a vaccine that would stimulate the production of antibodies capable of binding to and neutralizing two types of virus at once. Although the Zika virus was not previously thought to be dangerous, neurological complications including Guillain-Barré syndrome have been observed in Zika patients in Brazil and French Polynesia. This virus is also the cause of severe fetal brain development defects (microcephaly), resulting in irreversible intellectual disability. "The antibodies could be used, for example, to protect pregnant women at risk of contracting the Zika virus, because there is currently no vaccine or treatment for this disease," concluded Félix Rey.

This work was funded by the institutions mentioned above and by the European Framework Programme (FP7) DENFREE, IBEID LabEx (Integrative Biology of Emerging Infectious Diseases) and the FlaviStem grant (ANR / FWF).

[1] Involved laboratories:
Virology (CNRS/Institut Pasteur)
Structural virology (CNRS/Institut Pasteur).

http://www.eurekalert.org/pub_releases/2016-06/ehs-hpd062316.php

Health-care providers do not fully understand cancer risk from CT scans

Knowledge of radiation dose and associated risks varies among referring physicians, radiologists, and technicians, according to a new study in the Journal of Medical Imaging and Radiation Sciences

Philadelphia, PA, - Computed tomography (CT) scans are an invaluable diagnostic tool in modern medicine, but they do come at a price: exposing patients to potentially dangerous ionizing radiation. Doctors and other healthcare professionals may not be fully aware of a CT scan's effect on lifetime malignancy risk. A new study in the Journal of Medical Imaging and Radiation Sciences surveyed doctors, radiologists, and imaging technologists regarding their beliefs about radiation exposure from CT. The survey found that while most respondents recognized there is an increased risk of cancer from CT, many underestimated the actual radiation dose.

Researchers from the University of Saskatchewan wanted to assess healthcare providers' knowledge regarding radiation dosing from CT scans. Using a survey of medical professionals in Saskatchewan, investigators found that 73% of physicians, 97% of radiologists, and 76% of technologists correctly identified that there is an increased cancer risk from one abdominal-pelvic CT. However, only 18% of physicians, 28% of radiologists, and 22% of technologists were able to correctly identify the dose in relation to chest x-rays. Although 48% of physicians, 78% of radiologists and 63% of technologists either accurately estimated or overestimated this dose, many respondents underestimated the dose level.

"Underestimating radiation dose from a CT scan is more concerning than knowing the exact dose level, particularly when it is a vast underestimation, as this may lead to minimization of the risk estimate when considering a test," explained lead investigator David Leswick, MD, FRCPC, Department of Medical Imaging, College of Medicine, University of Saskatchewan (Saskatoon, Saskatchewan).

The issue of radiation exposure is significant as doctors continue to order CT scans with increasing frequency. In Canada alone, there were an estimated 4.4 million CT scans conducted in 2011-2012. Measured in millisieverts (mSv), the average radiation dose from an abdominal-pelvic CT is 10 mSv, compared to 0.02 to 0.2 mSv from one chest x-ray, meaning that a radiation dose from a CT scan is best approximated as between that from 100-250 chest radiographs.

"Although risk from radiation dose levels in the range of medical imaging procedures is small, it is real as evidenced from atomic bomb survivors and nuclear industry workers showing significantly increased risk of malignancy after exposure to doses in the range of diagnostic CT," said Dr. Leswick. "The risk of fatal malignancy may be as high as 1 in 1000 for a 10-mSv exposure (approximate dose of an abdomen-pelvis CT). This risk is significant on a population basis, with up to 2% of cancers in the United States population possibly attributable to CT."

With such a clear risk relationship between radiation exposure and cancer, it is imperative that healthcare providers understand the facts to ensure the benefits outweigh the possible danger when ordering a diagnostic CT. The survey indicated that 93% of respondents were interested in radiation dose feedback when considering ordering a CT scan. Automated dose calculation software and radiology information systems can be integrated into electronic ordering, which would give doctors immediate access to information when considering ordering a scan.

Another interesting aspect highlighted by the survey was some confusion regarding radiation exposure from magnetic resonance imaging (MRI) and ultrasound. MRIs and ultrasounds do not employ ionizing radiation and yet 20% of physicians, 6% of radiologists, and 7% of technologists attributed radiation exposure to MRIs and 11% of physicians, 0% of radiologists, and 7% of technologists believed an ultrasound used radiation. "Belief that ionizing radiation is utilized by ultrasound and MRI is troubling as it may result in underutilization of these imaging modalities because of unfounded radiation concerns," added Dr. Leswick.

While CT scans can be a lifesaving diagnostic tool, they also present a potential danger if they are overused or incorrectly implemented. It is vital that doctors and other healthcare practitioners fully understand the implications of ordering a CT scan and that patients are counseled appropriately about all available forms of testing and the potential radiation exposure involved.

"Unfortunately, healthcare providers including physicians, radiologists, and medical imaging technologists are often not aware of radiation doses for common CT scans," concluded Dr. Leswick. "It is important for healthcare professionals (including referring physicians, radiologists, and technologists) to be aware of radiation dose levels and risks from imaging tests for several reasons, including the ability to weigh the risks and benefits of tests, counsel patients on relevant risks, optimize protocols to minimize radiation dose, and select appropriate protocols to minimize radiation dose."

http://www.eurekalert.org/pub_releases/2016-06/cifs-vqq062316.php

Volcanoes get quiet before they erupt!

Periods of seismic quiet occur immediately before eruptions

Washington, D.C.-- When dormant volcanoes are about to erupt, they show some predictive characteristics--seismic activity beneath the volcano starts to increase, gas escapes through the vent, or the surrounding ground starts to deform. However, until now, there has not been a way to forecast eruptions of more restless volcanoes because of the constant seismic activity and gas and steam emissions. Carnegie volcanologist Diana Roman, working with a team of scientists from Penn State, Oxford University, the University of Iceland, and INETER* has shown that periods of seismic quiet occur immediately before eruptions and can thus be used to forecast an impending eruption for restless volcanoes. The duration of the silence can indicate the level of energy that will be released when eruption occurs. Longer quiet periods mean a bigger bang. The research is published in Earth and Planetary Science Letters.

The team monitored a sequence of eruptions at the Telica Volcano in Nicaragua in 2011. It is a so-called stratovolcano, with a classic-looking cone built up by many layers of lava and ash. They started monitoring Telica in 2009 with various instruments and by 2011 they had a comprehensive network within 2.5 miles (4 kilometers) of the volcano's summit.

The 2011 eruptive event was a month-long series of small to moderate ash explosions. Prior to the eruption, there was a lack of deep seismicity or deformation, and small changes in sulfur dioxide gas emissions, indicating that the eruption was not driven by fresh magma. Instead, the eruption likely resulted from the vents being sealed off so that gas could not escape. This resulted in an increase in the pressure that eventually caused the explosions.

Of the 50 explosions that occurred, 35 had preceding quiet periods lasting 30 minutes or longer. Thirteen explosions were preceded by quiet intervals of at least five minutes. Only two of the 50 did not have any quiet period preceding the explosion. 6"It is the proverbial calm before the storm," remarked Roman. "The icing on the cake is that we could also use these quiet periods to forecast the amount of energy released."

The researchers did a "hindsight" analysis of the energy released. They found that the longer the quiet phase preceding an explosion, the more energy was released in the ensuing explosion. The quiet periods ranged from 6 minutes before an explosion to over 10 hours (619 minutes) for the largest explosion.

The researchers were also able to forecast a minimum energy for impending explosions based on the data from the previous quiet/explosion pairs and the duration of the particular quiet period being analyzed. The correlation between duration of quiet periods and amount of energy released is tied to the duration of the gas pathways being blocked. The longer the blockage, the more pressure builds up resulting in more energy released. Sealing might be occurring due to mineral precipitation in cracks that previously acted as gas pathways, or due to the settling of the rock near the volcano's surface.

"What is clear is that this method of careful monitoring of Telica or other similar volcanoes in real time could be used for short-term forecasts of eruptions," Roman said. "Similar observations of this phenomenon have been noted anecdotally elsewhere. Our work has now quantified that quiet periods can be used for eruption forecasts and that longer quiet periods at recently active volcanoes could indicate a higher risk of energetic eruptions."

**The paper's other authors are Mel Rodgers of Oxford University, Peter LaFemina of Penn State University, Halldor Geirsson of the University of Iceland, and Virginia Tenorio of the Instituto Nicaraguense de Estudios Territoriales.*

This work was supported by the National Science Foundation and the Nicaraguan Institute of Earth Sciences (INETER).

http://www.eurekalert.org/pub_releases/2016-06/vua-ewc062416.php

Eyewitnesses who collaborate make fewer mistakes in police interview

Witnesses correct each other's errors.

Two recently published research studies by legal psychologists Annelies Vredeveldt and Peter van Koppen at Vrije Universiteit Amsterdam show that witnesses make fewer errors when they are interviewed together than when they are interviewed separately. This stands in sharp contrast with current police guidelines to always interview witnesses separately.

In a unique collaboration between VU Amsterdam and the Toneelschuur theatre in Haarlem, in the first study the researchers asked attendees of an emotional play to testify about a rape-and-murder scene they saw a week earlier.

Witnesses were interviewed either in pairs or alone. Two witnesses who were interviewed together reported the same amount of information but made substantially fewer errors than two witnesses interviewed separately.

In the second study, the researchers interviewed a larger number of witnesses (80 people) and found the same result.

These findings are surprising in light of previous legal psychological research showing that witnesses can contaminate each other's memory. The difference is that in those studies, artificial ways were used to increase errors, such as letting the witness talk to a 'fake witness' who purposefully introduced errors in the discussion. In contrast, the research by VU Amsterdam investigated naturalistic discussions between witnesses.

Eyewitness pairs with more effective communication styles remembered more

Moreover, the research showed that couples with an effective communication style remembered more together than couples who communicated less effectively. The research shows that the best way of collaborating is to repeat or rephrase your partner's statements and then elaborate by adding extra information.

"The research findings show that collaboration between witnesses can also have benefits", the researchers said.

"Until now, most people assumed that discussion between witnesses has only disadvantages." Vredeveldt and Van Koppen expect that this research will lead to widespread interest from both academics and police practitioners.

The research has been published in two prestigious international academic journals: the first study in Memory and the second study in Legal and Criminological Psychology.

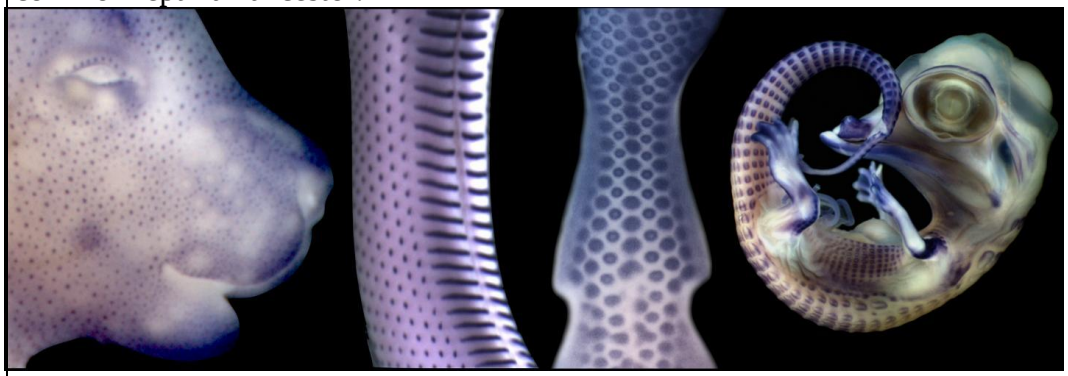
The studies form part of the research conducted by the Amsterdam Laboratory of Legal Psychology (ALLP).

http://www.eurekalert.org/pub_releases/2016-06/udq-hfa062016.php

Hairs, feathers and scales have a lot in common!

The potential evolutionary link between hairs in mammals, feathers in birds and scales in reptiles has been debated for decades.

Today, researchers of the University of Geneva (UNIGE) and the SIB Swiss Institute of Bioinformatics, Switzerland, demonstrate that all these skin appendages are homologous: they share a common ancestry. On the basis of new analyses of embryonic development, the Swiss biologists evidenced molecular and micro-anatomical signatures that are identical between hairs, feathers and scales at their early developmental stages. These new observations, published today in Science Advances, indicate that the three structures evolved from their common reptilian ancestor.



Placodes (spots stained in dark blue by the expression of an early developmental gene) are visible before the development of hair, scales and feathers in (from left to right) the mouse, the snake, the chicken and the crocodile. **UNIGE 2016 (Tzika, Di-Poï, Milinkovitch).**

Mammalian hairs and avian feathers develop from a similar primordial structure called a 'placode': a local thickening of the epidermis with columnar cells that reduce their rate of proliferation and express very specific genes. This observation has puzzled evolutionary and developmental biologists for many years because birds and mammals are not sister groups: they evolved from different reptilian lineages. According to previous studies, reptiles' scales however do not develop from an anatomical placode. This would imply that birds and mammals have independently 'invented' placodes during their evolution.

The single evolutionary origin of placodes revealed

In 2015, a team from Yale University (USA) published an article showing that scales, hairs and feathers share molecular signatures during their development. These results fueled an old debate between two schools. One defends that these molecular signatures suggest a common evolutionary origin of skin appendages,

whereas the other proposes that the same genes are re-used for developing different skin appendages.

Today, Nicolas Di-Poï and Michel C. Milinkovitch at the Department of Genetics and Evolution of the UNIGE Faculty of Science and at the SIB put this long controversy to rest by demonstrating that scales in reptiles develop from a placode with all the anatomical and molecular signatures of avian and mammalian placodes. The two scientists finely observed and analysed the skin morphological and molecular characteristics during embryonic development in crocodiles, snakes and lizards. 'Our study not only provides new molecular data that complement the work of the American team but also reveals key microanatomical facts, explains Michel Milinkovitch. Indeed, we have identified in reptiles new molecular signatures that are identical to those observed during the development of hairs and feathers, as well as the presence of the same anatomical placode as in mammals and birds. This indicates that the three types of skin appendages are homologous: the reptilian scales, the avian feathers and the mammalian hairs, despite their very different final shapes, evolved from the scales of their reptilian common ancestor.'

A key gene for skin appendage development

During their new study, the researchers from UNIGE and SIB also investigated the bearded dragon, a species of lizard that comes in three variants. The first is the normal wild-type form. The second has scales of reduced size because it bears one copy of a natural genetic mutation. The third has two copies of the mutation ... and lacks all scales. By comparing the genome of these three variants, Di-Poï and Milinkovitch have discovered the gene affected by this mutation. 'We identified that the peculiar look of these naked lizards is due to the disruption of the ectodysplasin-A (EDA), a gene whose mutations in humans and mice are known to generate substantial abnormalities in the development of teeth, glands, nails and hairs', says Michel Milinkovitch. The Swiss researchers have demonstrated that, when EDA is malfunctioning in lizards, they fail to develop a proper scale placode, exactly as mammals or birds affected with similar mutations in that same gene cannot develop proper hairs or feathers placodes. These data all coherently indicate the common ancestry between scales, feathers and hairs.

The next challenge for the Swiss team, and many other researchers around the world, is to decipher the fine mechanisms explaining the diversity of forms of skin appendages. How has the ancestral scaly skin given rise to the very different morphologies of scales, feathers and hairs, as well as the astonishing variety of forms that these appendages can take?

These future studies will hopefully fine-tune our understanding of the physical and molecular mechanisms generating the complexity and the diversity of life during evolution.

http://www.eurekalert.org/pub_releases/2016-06/luhs-ndc062416.php

New devices causing 'paradigm shift' in stroke care

New devices called stent retrievers, which effectively reverse strokes, have revolutionized the treatment of certain stroke patients, according to an article in the journal Expert Review of Neurotherapeutics.

MAYWOOD, IL - "Stent retrievers are a major advance in acute ischemic stroke care and will have significant impact on the evolution of stroke systems of care," according to the article by Loyola Medicine neurologists Rick Gill, MD and Michael J. Schneck, MD. Dr. Gill is the outgoing chief resident and Dr. Schneck is a professor in the Department of Neurology of Loyola University Chicago Stritch School of Medicine.

Eighty-seven percent of strokes are ischemic, meaning they are caused by clots that block blood flow to a portion of the brain. In selected patients, stent retrievers can be used to remove such clots. Loyola used stent retrievers on 34 patients in 2015, and 21 patients during the first six months of 2016.

A stent retriever is a self-expanding mesh tube attached to a wire, which is guided through a catheter (thin tube). The endovascular specialist inserts the catheter in an artery in the groin and guides the catheter through various blood vessels all the way up to the brain.

Once the stent retriever reaches the blockage, the endovascular specialist deploys it. The device pushes the gelatinous blood clot against the wall of the blood vessel, immediately restoring blood flow. The stent retriever then is used to grab the clot, which is pulled out when the surgeon removes the catheter.

"With the advent of stent retriever devices, there has been a paradigm shift in the utilization of endovascular therapies for acute ischemic stroke," Drs. Gill and Schneck write. (Endovascular refers to catheter-based surgery.)

Drs. Gill and Schneck describe how the current generation of stent retrievers, including the TREVO and Solitaire devices, are a remarkable improvement over earlier devices such as MERCI and Penumbra that employed different technology. Studies of these earlier devices showed results that were equivocal at best. But more recent trials of stent retrievers consistently show the newer devices are clearly superior to the intravenous drug tissue plasminogen activator (tPA) alone in reducing disability from strokes.

The clot-busting drug tPA can restore blood flow and limit stroke damage, if it is given within 4.5 hours of the onset of the stroke and the clot is small enough. (If the patient is older than 80, the cutoff time is three hours.) But in many patients, tPA either would not be safe to take, or would not be sufficient by itself to restore blood flow. In such patients, stent retrievers often can be used to remove the clot.

Drs. Gill and Schneck foresee future device improvements that will do an even better job of restoring blood flow and increasing the number of patients who could benefit.

Stent retrievers also will affect where stroke patients are treated. Paramedics will play an important role in routing higher severity stroke patients, who could benefit from stent retrievers, to centers that have the capability to perform neuroendovascular procedures, Drs. Gill and Schneck write.

http://bit.ly/28YEjNN

China Planning Underwater Great Wall of Robots

Two recent and interconnected developments out of China suggest that the world's most populous nation has big plans for the deep seas.

By Glenn McDonald, Discovery News

China is designing a manned deep-sea "space station" to hunt for mineral resources in the South China Sea, according to a recent Bloomberg report. The proposed location is significant for a couple of reasons. For one thing, the South China Sea is a highly disputed area these days among China and its neighbors, especially Vietnam and the Philippines.

Vertically speaking, the location is also pretty remarkable for different reasons. The oceanic base would be built as deep as 3,000 meters (9,800 feet) below the surface, and would be inhabited by a full time crew. No one has ever attempted to build a manned underwater station at those depths.

But more worrisome for international observers is the idea that the underwater station could serve as an anchor for China's other big deep-sea initiative, the so-called Underwater Great Wall of China.

According to reports that surfaced in May, the underwater wall refers to a network of floating and submerged sensors designed to detect enemy submarines. Intelligence agencies have presumably known about these plans for a while, but China has only recently gone public with details on the system -- and the underwater robots and drones that would be involved.

In a recent military exhibit, Chinese government officials showed off a fleet of unmanned vehicles -- a.k.a. sea drones -- that would be part of the underwater wall defense system. These drones would be capable of maneuvering both on the surface of the water and at various depths beneath the waves. The sea drones would also be capable of carrying anti-submarine weapons and other payloads. The image above shows a kind of diorama exhibit from the Chinese presentation.

Back to the South China Sea space station: While the manned underwater base would be chiefly used for natural resource development, the platform will also be movable and could be used for military purposes, said Chinese officials at yet another recent presentation.

China's plans -- for a deep-sea space station and an underwater wall of drones -- should keep military strategists around the world busy for the next few years. On the more hopeful side, both projects could have massive potential for the advancement of undersea scientific projects.

http://www.eurekalert.org/pub_releases/2016-06/osu-asb062016.php

A shampoo bottle that empties completely -- every last drop

Coating to make soap pour cleanly out of plastic bottles, reduce waste and frustration

COLUMBUS, Ohio--It's one of life's little annoyances: that last bit of shampoo that won't quite pour out of the bottle. Or the last bit of hand soap, or dish soap, or laundry detergent.

Now researchers at The Ohio State University have found a way to create the perfect texture inside plastic bottles to let soap products flow freely. They describe the patent-pending technology in a paper to appear in the journal Philosophical Transactions of the Royal Society on June 27.

The technique involves lining a plastic bottle with microscopic y-shaped structures that cradle the droplets of soap aloft above tiny air pockets, so that the soap never actually touches the inside of the bottle. The "y" structures are built up using much smaller nanoparticles made of silica, or quartz--an ingredient in glass--which, when treated further, won't stick to soap.

If it sounds like engineers Bharat Bhushan and Philip Brown went to a lot of trouble to solve this problem, you're right. But the solution they found is actually simpler and less expensive than alternatives under development elsewhere. And it works for a common plastic used to package foodstuffs and household goods: polypropylene.

"It's what you'd call a first-world problem, right? 'I can't get all of the shampoo to come out of the bottle.' But manufacturers are really interested in this, because they make billions of bottles that end up in the garbage with product still in them," said Bhushan, Ohio Eminent Scholar and Howard D. Winbigler Professor of mechanical engineering at Ohio State. Coatings already exist to help food, but not soap, pour out of their containers, he said.

"Compared to soaps, getting ketchup out of a bottle is trivial. Our coating repels liquids in general, but getting it to repel soap was the hard part."

The key, he explained, is surface tension--the tendency of the molecules of a substance to stick to each other. Ketchup and other sauces are made mostly of water, and water molecules tend to stick to each other more than they stick to plastic.

But surfactants--the organic molecules that make soap "soapy"--are just the opposite: They have a very low surface tension and stick to plastic easily,

explained Brown, a postdoctoral fellow. "It was an extra challenge for us to make a surface that could repel surfactant," he agreed.

Their goal, which was suggested by a commercial shampoo manufacturer, was to create a shampoo bottle lining that was cheap, effective and environmentally friendly.

Soap and shampoo clean our skin and hair by bonding chemically with both oil and water, so the surface oils that were on our bodies wash off when we rinse. The same goes for dishes. During clothes washing, detergent performs double-duty, releasing oils and also helping water penetrate fabrics. It's that tenacity that makes the last drops of surfactant cling to the insides of bottles.

Bhushan and Brown came up with a method to spray-coat a small amount of solvent and ultra-fine silica nanoparticles onto the inside of bottles. Manufacturers already use solvents to change the texture of molded plastics, because they cause the surface of the plastic to soften a little. By mixing the silica and solvent, the researchers were able to soften the surface of the polypropylene just enough that when the plastic re-hardened, the silica would be embedded in the surface.

The structures are only a few micrometers--millionths of a meter--high, and covered in even smaller branchlike projections. They look like shaggy heart-shaped pillows, but they're hard as glass.

They don't cover the inside of the bottle completely, either, but instead are planted a few micrometers apart. The main branches of the "y" overhang the plastic surface at an angle less than 90 degrees--steep enough that water, oils and even surfactant can't physically sustain a droplet shape that would fall in between the branches and touch the plastic. "You end up with air pockets underneath, and that's what gives you liquid repellency," Brown said. Instead of spreading out on the surface, the soap droplets form beads and roll right off.

Researchers have known for some time that a support structure with the right angle of overhang would solve this problem, and some have tried to carve the shapes into plastic manually using photolithography--the same technique that shapes computer chips.

"That's expensive and time consuming," Brown said. "Plus, they end up with fragile little overhangs that snap off. We embedded a hard material directly into the polymer surface, so we know it's durable."

Polypropylene isn't the most common plastic bottle material, but 177 million pounds of it were made into bottles and bottle lids in the United States in 2014 alone, according to the American Chemistry Council (ACC). Aside from shampoo, soap and detergent bottles, it's also used for yogurt tubs, ketchup bottles and medical bottles, single-serve coffee pods and Starbucks iced beverage cups.

Polypropylene is classified as a "number 5" plastic by the Resin Identification Coding System. A recent ACC report found that recycling of number 5 plastics is on the rise, increasing from 44.2 million pounds in 2013 to 45.6 million pounds in 2014. Only about two-thirds of American curbside recycling services accept it, but commercial companies such as Preserve of Waltham, Massachusetts, and Whole Foods grocery stores nationwide are working with manufacturers and retailers to collect number 5 plastic containers and make them into useful products.

The Ohio State invention could actually aid recycling. Before plastic bottles can be recycled, they have to be rinsed completely clean, and Bhushan suspects that he's not the only person who doesn't bother.

"We all struggle with shampoo bottles at home," Bhushan said. "I have a few in my shower right now. Trying to get the last drop out, I put it upside down, and my wife adds water to the bottle and fights with it for a while, and then we give up and just throw it away."

With further development, the university hopes to license the coating technique to manufacturers--not just for shampoo bottles, but for other plastic products that have to stay clean, such as biomedical devices or catheters. They have already applied the same technique to polycarbonate, a plastic used in car headlights and smartphone cases, among other applications.

http://www.eurekalert.org/pub_releases/2016-06/gc-rcf062316.php

Radioactive cesium fallout on Tokyo from Fukushima concentrated in glass microparticles

Radioactive fallout after the Fukushima accident was concentrated and deposited in non-soluble glass microparticles, as a type of 'glassy soot'

New research shows that most of the radioactive fallout which landed on downtown Tokyo a few days after the Fukushima accident was concentrated and deposited in non-soluble glass microparticles, as a type of 'glassy soot'. This meant that most of the radioactive material was not dissolved in rain and running water, and probably stayed in the environment until removed by direct washing or physical removal. The particles also concentrated the radioactive caesium (Cs), meaning that in some cases dose effects of the fallout are still unclear. These results are announced at the Goldschmidt geochemistry conference in Yokohama, Japan.

The flooding of the Fukushima Daiichi Nuclear Power Plant (FDNPP) after the disastrous earthquake on March 11 2011 caused the release of significant amounts of radioactive material, including caesium (Cs) isotopes 134Cs (half-life, 2 years) and 137Cs (half-life, 30 years).

Japanese geochemists, headed by Dr Satoshi Utsunomiya (Kyushu University, Japan), analysed samples collected from within an area up to 230 km from the FDNPP. As caesium is water-soluble, it had been anticipated that most of the radioactive fallout would have been flushed from the environment by rainwater. However, analysis with state-of-the-art electron microscopy in conjunction with autoradiography techniques showed that most of the radioactive caesium in fact fell to the ground enclosed in glassy microparticles, formed at the time of the reactor meltdown.

The analysis shows that these particles mainly consist of Fe-Zn-oxides nanoparticles, which, along with the caesium were embedded in Si oxide glass that formed during the molten core-concrete interaction inside the primary containment vessel in the Fukushima reactor units 1 and/or 3. Because of the high Cs content in the microparticles, the radioactivity per unit mass was as high as ~4.4x1011 Bq/g, which is between 107 and 108 times higher than the background Cs radioactivity per unit mass of the typical soils in Fukushima.

Closer microparticle structural and geochemical analysis also revealed what happened during the accident at FDNPP. Radioactive Cs was released and formed airborne Cs nanoparticles. Nuclear fuel, at temperatures of above 2200 K (about as hot as a blowtorch), melted the reactor pressure vessel resulting in failure of the vessel. The airborne Cs nanoparticles were condensed along with the Fe-Zn nanoparticles and the gas from the molten concrete, to form the SiO2 glass nanoparticles, which were then dispersed.

Analysis from several air filters collected in Tokyo on 15 March 2011 showed that 89% of the total radioactivity was present as a result of these caesium-rich microparticles, rather than the soluble Cs, as had originally been supposed.

According to Dr Satoshi Utsunomiya;

"This work changes some of our assumptions about the Fukushima fallout. It looks like the clean-up procedure, which consisted of washing and removal of top soils, was the correct thing to do. However, the concentration of radioactive caesium in microparticles means that, at an extremely localised and focused level, the radioactive fallout may have been more (or less) concentrated than anticipated. This may mean that our ideas of the health implications should be modified".

Commenting, Prof. Bernd Grambow, Director of SUBATECH laboratory, Nantes, France and leader of the research group on interfacial reaction field chemistry of the ASRC/JAEA, Tokai, Japan, said:

"The leading edge observations by nano-science facilities presented here are extremely important. They may change our understanding of the mechanism of long range atmospheric mass transfer of radioactive caesium from the reactor accident at Fukushima to Tokyo, but they may also change the way we assess inhalation doses from the caesium microparticles inhaled by humans. Indeed, biological half- lives of insoluble caesium particles might be much larger than that of soluble caesium".

NOTE: the headline of this press release was shortened to comply with Eurekalert restrictions. The original was "Most radioactive caesium fallout on Tokyo from Fukushima accident was concentrated in glass microparticles"

http://bit.ly/28VdLsq

Mammal fossil skeleton from dinosaur era found in Japan

First discovery of an entire mammal from the dinosaur era in Japan

A fossil skeleton that forms almost the whole body of a herbivorous mammal has been discovered from a Lower Cretaceous layer of earth of about 120 million years ago in Katsuyama, Fukui Prefecture, central Japan, it was announced Saturday.

It is rare that a fossil of a whole mammal from the dinosaur era is found, and this is the first such discovery in Japan, according to the Fukui Prefectural Dinosaur Museum and Fukui Prefectural University, which announced the finding.

The fossil skeleton was discovered in June 2014 by Kakeru Funato, then an elementary school fourth-grade boy, who was attending a fossil excavation event in the Katsuyama Dinosaur Forest Park.

A computerized tomography examination by the museum and the university found that it is the skeleton of a small grass-eating mammal from an extinct group called Multituberculata, which has similar characteristics to Rodentia.

http://www.eurekalert.org/pub_releases/2016-06/uom-ron062216.php

Researchers open new path of discovery in Parkinson's disease

Neuron cell death may be caused by overactive immune system

A team of scientists led by Dr. Michel Desjardins from the University of Montreal and Dr. Heidi McBride from the Montreal Neurological Institute and Hospital (MNI) at McGill University have discovered that two genes associated with Parkinson's disease (PD) are key regulators of the immune system, providing direct evidence linking Parkinson's to autoimmune disease.

Using both cellular and mouse models, the team has shown that proteins produced by the two genes, known as PINK1 and Parkin, are required to prevent cells from being detected and attacked by the immune system.

When PINK1 and Parkin are dysfunctional, as is the case in a subset of Parkinson's patients, cells display small parts of proteins at their surface, known as antigens, derived from mitochondria. The presence of these antigens at the cell surface causes the activation of immune cells called lymphocyte T cells. These T cells, which can enter the brain, have the ability to destroy any cell displaying the mitochondrial antigens on their surface.

Parkinson's is caused by the death of dopamine-producing neurons in the brain. An overactive immune system due to dysfunctional PINK1 and Parkin genes could explain why dopaminergic neurons die in Parkinson's patients. This indicates that Parkinson's may be one of many autoimmune diseases, including multiple sclerosis, Type 1 diabetes, rheumatoid arthritis, and lupus. An autoimmune disease is one in which the body's own immune system attacks healthy cells.

Researchers suspected that mitochondria, organelles within cells that are responsible for the production of energy and other metabolites, play a role in Parkinson's. It was widely believed that mitochondria become damaged in Parkinson's patients, creating a toxic build-up of broken mitochondria that eventually leads to neuron cell death. However, it has been difficult to provide evidence that this is effectively happening in animal models.

The new findings of the Desjardins/McBride teams linking PD to autoimmune mechanisms, published in the prestigious journal Cell on June 23, have been validated in a mouse model of Parkinson's disease where PINK1 or Parkin are absent.

"Clinicians have shown that the immune system is activated in the brain of PD patients," says Dr. Diana Matheoud, a postdoctoral fellow from the University of Montreal and the article's first author. "Our study explains how an attack by the immune system may be responsible for the destruction of dopaminergic neurons during the disease. We are currently testing whether autoimmune mechanisms lead to the loss of dopaminergic neurons in mice, and developing systems to extend our study to human neurons."

"Antigen presentation was not believed to play a direct role in Parkinson's disease," says McBride. "While most laboratories are following the trail of the 'toxic mitochondria' model, our path led us to observe Parkinson's disease from a different point of view. Our approach, centered on the immune system, led us down a different road where we were able to observe that autoimmunity is likely to play an important role in the progression of the disease."

Now that a link has been established between two key genes involved in the pathology of Parkinson's disease and autoimmune mechanisms, the next step is to develop drugs that can limit the presentation of mitochondrial antigens. Remarkably, the mechanism by which mitochondrial antigens are presented involves a process of vesicle formation, originally described by the McBride group, offering molecular targets for the development of new drugs in an effort to block this process.

The researchers' findings may also lead to better treatments for other diseases. "We think that our study is paradigm shifting because we have identified a new biological pathway linking mitochondria to immune mechanisms in Parkinson's disease. This opens the possibility to use therapies based on modulation of the immune system, something already done for the treatment of other diseases," says Desjardins. "Interestingly, the role played by PINK1 and Parkin in limiting the presentation of mitochondrial antigens may not only regulate a process that impact Parkinson's disease, but may also affect other autoimmune diseases like diabetes and lupus, and primary biliary cirrhosis, where a link to mitochondrial antigen presentation has been observed."

"This paper suggests an entirely novel mechanism by which these recessive, inherited mutations may lead to neurodegeneration," says Jon Stoessl, Professor and Head of Neurology at the University of British Columbia & Vancouver Coastal Health, and former Director of the Pacific Parkinson's Research Centre. "There has been much interest in the potential role of inflammation in PD. Previous studies on Parkin and PINK1 have focused on disruption of mitochondrial housekeeping functions. While the current findings may clearly be related, they suggest an entirely novel approach to the development of targeted therapies. It should be remembered that these are rare causes of Parkinson's disease and the relevance to dominantly inherited and sporadic forms of disease remains to be determined."

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