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Exploding star missing from formation of solar system

A new study published by University of Chicago researchers challenges the notion that the force of an exploding star forced the formation of the solar system.

In this study, published online last month in Earth and Planetary Science Letters, authors Haolan Tang and Nicolas Dauphas found the radioactive isotope iron 60 - the telltale sign of an exploding star - low in abundance and well mixed in solar system material. As cosmochemists, they look for remnants of stellar explosions in meteorites to help determine the conditions under which the solar system formed.

Some remnants are radioactive isotopes: unstable, energetic atoms that decay over time. Scientists in the past decade have found high amounts of the radioactive isotope iron 60 in early solar system materials. "If you have iron 60 in high abundance in the solar system, that's a 'smoking gun' - evidence for the presence of a supernova," said Dauphas, professor in geophysical sciences.

Iron 60 can only originate from a supernova, so scientists have tried to explain this apparent abundance by suggesting that a supernova occurred nearby, spreading the isotope through the explosion.

But Tang and Dauphas' results were different from previous work: They discovered that levels of iron 60 were uniform and low in early solar system material. They arrived at these conclusions by testing meteorite samples. To measure iron 60's abundance, they looked at the same materials that previous researchers had worked on, but used a different, more precise approach that yielded evidence of very low iron 60.

Previous methods kept the meteorite samples intact and did not remove impurities completely, which may have led to greater errors in measurement. Tang and Dauphas' approach, however, required that they "digest" their meteorite samples into solution before measurement, which allowed them to thoroughly remove the impurities. This process ultimately produced results with much smaller errors. "Haolan has dedicated five years of very hard work to reach these conclusions, so we did not make those claims lightly. We've been extremely careful to reach a point where we're ready to go public on those measurements," Dauphas said.

To address whether iron 60 was widely distributed, Tang and Dauphas looked at another isotope of iron, iron 58. Supernovae produce both isotopes by the same processes, so they were able to trace the distribution of iron 60 by measuring the distribution of iron 58. "The two isotopes act like inseparable twins: Once we knew where iron 58 was, we knew iron 60 couldn't be very far away," Dauphas explained.

They found little variation of iron 58 in their measurements of various meteorite samples, which confirmed their conclusion that iron 60 was uniformly distributed. To account for their unprecedented findings, Tang and Dauphas suggest that the low levels of iron 60 probably came from the long-term accumulation of iron 60 in the interstellar medium from the ashes of countless stars past, instead of a nearby cataclysmic event like a supernova.

If this is true, Dauphas said, there is then "no need to invoke any nearby star to make iron 60." However, it is more difficult to account for the high abundance of aluminum 26, which implies the presence of a nearby star. Instead of explaining this abundance by supernova, Tang and Dauphas propose that a massive star (perhaps more than 20 times the mass of the sun) sheds its gaseous outer layers through winds, spreading aluminum 26 and contaminating the material that would eventually form the solar system, while iron 60 remained locked inside the massive star's interior. If the solar system formed from this material, this alternate scenario would account for the abundances of both isotopes. "In the future, this study must be considered when people build their story about solar system origin and formation," Tang said. - *Chelsea Leu*

Citation: "Abundance distribution, and origin of 60Fe in the solar protoplanetary disk," by Haolan Tang and Nicolas Dauphas, Earth and Planetary Science Letters, December 2012.

Funding: National Aeronautics and Space Administration, National Science Foundation, and the Packard Foundation.

http://www.eurekalert.org/pub_releases/2012-12/cu-csr121712.php

Chances seen rising for chikungunya outbreaks in NYC, Atlanta, Miami

Global travel and climate warming could be creating the right conditions for outbreaks of a new virus in this country, according to a new Cornell University computer model.

ITHACA, N.Y. – The model predicts that outbreaks of chikungunya, a painful virus transported by travelers and spread by the invasive Asian tiger mosquito, could occur in 2013 in New York City during August and September, in Atlanta from June through September, and year-round in Miami. The probability of a disease outbreak is correlated with temperature, as warmer weather allows the Asian tiger mosquito to breed faster and grow in numbers, according to the study published in the November issue of PLOS Neglected Tropical Diseases.

According to the simulation, there is a high probability of a chikungunya outbreak if a single infected person arrives in New York in July or August and is bitten by an Asian tiger mosquito. The risks are the same, but with wider time frames, for transmission in Atlanta and Miami, according to the paper.
Asian tiger mosquitoes were introduced to the United States in Texas in the 1980s; they are established up the East Coast into New Jersey and are rising in numbers in New York City. The aggressive mosquito outcompetes local varieties and transmits more than 20 pathogens, including chikungunya and dengue, said Laura Harrington, associate professor of entomology and the study's senior author.
"The virus is moving in people, and resident mosquito populations are picking it up," Harrington said.
The model estimates that with typical regional temperatures, a chikungunya outbreak in New York would infect about one in 5,000 people, said Diego Ruiz-Moreno, a postdoctoral associate and the paper's lead author.
"However, this number would increase drastically as temperatures rise due to climate change," Ruiz-Moreno said.
Chikungunya symptoms include a fever, severe joint pain, achiness, headache, nausea and fatigue, as well as "debilitating and prolonged" pain in the small joints of the hands and feet, according to the paper. The virus originated in Central Africa and is endemic in Southeast Asia. Since no chikungunya vaccine exists, U.S. residents can help prevent an outbreak by removing standing water, wearing long sleeves and repellent during the day when the mosquitoes feed, and knowing the risk and symptoms when traveling, Harrington said.
The study was funded by a National Institute for Food and Agriculture Hatch grant and Cornell's Atkinson Center for a Sustainable Future Climate Change and Disease Program.

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New type of cell division discovered

Research presented at ASCB annual meeting, Dec. 15-19, San Francisco

Researchers testing a theory about how a cell divides and how cancer begins have discovered a new type of cell division, the fundamental process by which cells replicate, Mark Burkard, MD, PhD, reported at the American Society for Cell Biology Annual Meeting, Dec. 17 in San Francisco.
The new type of cell division, which the researchers named klerokinesis, could be an evolutionary failsafe mechanism that could rescue a range of cell functions during embryonic development to genetic repair that can become abnormal if cell division fails to produce normal cells, said Dr. Burkard of the University of Wisconsin, Madison. "We hope to learn how to promote klerokinesis to help prevent cancer," he added.
Numerous cell divisions occur once a single egg is fertilized. The number of mitotic cycles is estimated at ~10^{13}, about 25 times the number of stars in our galaxy, according to Dr. Burkard. Beyond number, accuracy also counts in mitosis and was an early concern of Theodor Boveri, PhD, the German cell biology pioneer who in 1914 published the hypothesis that mistakes that occur when chromosomes divide during the mitotic cell cycle would lead to aneuploidy, characterized by too many or too few chromosomes in the resulting daughter cells. Such abnormal cells, Dr. Boveri believed, would lead to the out-of-control cell division that is a hallmark of cancer.
During the almost 100 years since Dr. Boveri's hypothesis was published, cell biologists have found considerable evidence that aneuploidy can promote the transformation of normal cells into cancer. Until the recent arrival of live single-cell videomicroscopy techniques, Dr. Burkard explained, it wasn't possible to examine a key feature of Boveri's theory: that aneuploidy results from the failure of cytokinesis, the separation of the cell membrane between segregated chromosomes to form two daughter cells.
Dr. Burkard and Wisconsin colleagues allowed human retinal pigment epithelial (RPE) cells in the lab to undergo karyokinesis, the orderly separation of chromosomes in mitosis, but blocked cytokinesis, the actual division into two daughter cells. This process resulted in single cells with two nuclei, termed binucleate. Binucleate cells were allowed to go through more mitotic cycles and proliferate.
The researchers said that they were surprised that one-third of the binucleate cells generated healthy daughter colonies. In addition, the majority of their progeny had chromosome sets that perfectly matched the original first-generation RPE cell. "This made Boveri's random chromosome assortment seem rather improbable," Dr. Burkard explained, "so we carefully observed the cells by time-lapse microscopy to see how this might be occurring."
They discovered that cells with two nuclei got stuck in the first growth phase of the cell cycle long enough for them to stretch apart, dividing the cytoplasm into two cells, each with its own nucleus. The process neatly preserved the accurately separated chromosome sets. Further work showed that these cells managed to divide without the usual proteins required for division of cell membranes in cytokinesis.
"We concluded that we were observing a new type of cell division," Dr. Burkard said, "which we term 'klerokinesis.' Klerokinesis is derived from the Greek root for allotted inheritance, chosen because each

daughter inherits a full set of chromosomes. Klerokinesis is a primitive mechanism of cell division that appears to be preserved in humans, as similar divisions have been observed in organisms such as slime molds, he added.
The American Cancer Society, Mary Kay Foundation, and National Institutes of Health funded this research.

http://www.eurekalert.org/pub_releases/2012-12/uom-bbc121712.php

Bullying by childhood peers leaves a trace that can change the expression of a gene linked to mood

Bullying among children may be a threat to their future mental health

A recent study by a researcher at the Centre for Studies on Human Stress (CSHS) at the Hôpital Louis-H. Lafontaine and professor at the Université de Montréal suggests that bullying by peers changes the structure surrounding a gene involved in regulating mood, making victims more vulnerable to mental health problems as they age.

The study published in the journal Psychological Medicine seeks to better understand the mechanisms that explain how difficult experiences disrupt our response to stressful situations. "Many people think that our genes are immutable; however this study suggests that environment, even the social environment, can affect their functioning. This is particularly the case for victimization experiences in childhood, which change not only our stress response but also the functioning of genes involved in mood regulation," says Isabelle Ouellet-Morin, lead author of the study.

A previous study by Ouellet-Morin, conducted at the Institute of Psychiatry in London (UK), showed that bullied children secrete less cortisol - the stress hormone - but had more problems with social interaction and aggressive behaviour. The present study indicates that the reduction of cortisol, which occurs around the age of 12, is preceded two years earlier by a change in the structure surrounding a gene (SERT) that regulates serotonin, a neurotransmitter involved in mood regulation and depression.

To achieve these results, 28 pairs of identical twins with a mean age of 10 years were analyzed separately according to their experiences of bullying by peers: one twin had been bullied at school while the other had not. "Since they were identical twins living in the same conditions, changes in the chemical structure surrounding the gene cannot be explained by genetics or family environment. Our results suggest that victimization experiences are the source of these changes," says Ouellet-Morin. According to the author, it would now be worthwhile to evaluate the possibility of reversing these psychological effects, in particular, through interventions at school and support for victims.

http://www.eurekalert.org/pub_releases/2012-12/asfc-ndi120312.php

Neurons die in Alzheimer's because of faulty cell cycle control before plaques and tangles appear

Research presented at ASCB annual meeting, Dec. 15-19, San Francisco

The two infamous proteins, amyloid-beta (Aβ) and tau, that characterize advanced Alzheimer's disease (AD), start healthy neurons on the road to cell death long before the appearance of the deadly plaques and tangles by working together to reactivate the supposedly blocked cell cycle in brain cells, according to research presented on Dec. 17 at the American Society for Cell Biology's Annual Meeting in San Francisco.

Working in a mouse model of AD, George Bloom, PhD, of the University of Virginia (UVA) reports that neurons in AD start dying because they break the first law of human neuronal safety – stay out of the cell cycle. Most normal adult neurons are permanently postmitotic; that is, they have finished dividing and are locked out of the cell cycle. In contrast, AD neurons frequently re-enter the cell cycle but fail to complete mitosis, and ultimately die. By considering this novel perspective on AD as a problem of the cell cycle, Dr. Bloom and colleagues at UVA and at the University of Alabama, Birmingham, have discovered what they call an "ironic pathway" to neuronal cell death. The process requires the coordinated action of both Aβ and tau, which are the building blocks of plaques and tangles, respectively. Dr. Bloom's results show just how toxic the two proteins can be even when free in solution and not aggregated into plaques and tangles.

Using mouse neurons grown in culture, the UVA researchers found that Aβ oligomers, which are small aggregates of just a few Aβ molecules each, induce the neurons to re-enter the cell cycle. Interestingly, the neurons must make and accumulate tau in order for this cell cycle re-entry to occur. The mechanism for this misplaced re-entry into the cell cycle requires that Aβ oligomers activate multiple protein kinase enzymes, each of which must then attach a phosphate to a specific site on the tau protein.

Following up on the cell culture results, Dr. Bloom and colleagues confirmed that Aβ-induced, tau-dependent cell cycle re-entry occurs in the brains of mice that were genetically engineered to mimic brains with human AD. The mouse brains were found to accumulate massive numbers of neurons that had transitioned from a permanent cell cycle stop, known as G0 (G zero), to G1, the first stage of the cell cycle, by the time they were 6

months old. Remarkably, otherwise identical mice that lacked functional tau genes showed no sign of cell cycle re-entry, confirming the cell culture results.
Neuronal cell cycle re-entry, a key step in the development of AD, can therefore be caused by signaling from Aβ through tau. Thus, Aβ and tau co-conspire to trigger seminal events in AD pathogenesis independently of their incorporation into plaques and tangles. Most important, Dr. Bloom believes that the activated protein kinases and phosphorylated forms of tau identified in this study represent potential targets for early diagnosis and treatment of AD.
This research was supported by the Alzheimer's Association (grant 4079 to GSB), Owens Family Foundation (GSB), NIH/NIGMS training grant T32 GM008136 that partially funded predoctoral training for two of the scientists, and NIH R01-NS075487 (EDR).

http://www.eurekalert.org/pub_releases/2012-12/osu-wlb121712.php

Who likes bling? The answer relates to social status

A desire for expensive, high-status goods is related to feelings of social status - which helps explain why minorities are attracted to bling, a new study suggests.

COLUMBUS, Ohio – Previous research had shown that racial minorities spend a larger portion of their incomes than do whites on conspicuous consumption – buying products that suggest high status. But a new study showed that whites could be induced to crave expensive, high-status products if they imagined themselves in a low-status position.
These findings cast doubt on the notion that urban minorities have developed a corrosive "bling culture" that is unique to them, said Philip Mazzocco, lead author of the study and assistant professor of psychology at Ohio State University's Mansfield campus. "Minorities don't buy high-status products because of some 'bling culture.' It is a basic psychological tendency that we all share when we're feeling inferior in some part of our life," Mazzocco said. "Anyone who is feeling low in status is going to try to compensate. And in our capitalistic, consumption-oriented society, one way to compensate is to buy high-status products." Mazzocco conducted the study with Derek Rucker, Adam Galinsky and Eric Anderson of Northwestern University. The findings appear in a recent issue of the Journal of Consumer Psychology.
For the study, the researchers conducted several related experiments. In the first experiment, 146 American adults – about half white and half black – were told they would be participating in a study of consumer preferences. They were asked to rate how positively or negatively they viewed 10 products on a nine-point scale from "extremely negative" to "extremely positive."
Five of the products had been rated by a separate group of people as high in status (fur coat, cuff links, caviar, an Italian suit and Italian loafers), while five were rated as relatively low in status (vacuum cleaner, sofa, refrigerator, washing machine and an unbranded shirt).
The study found that, overall, blacks had more positive evaluations of the high-status products than did whites. But more importantly, blacks who considered their race to be an important part of their identity rated high-status goods higher than did blacks who had lower racial identification. There was no such difference among whites in the study. "Because African Americans are seen as lower in status in our society, those who identify more strongly with being black are more likely to compensate by seeking high-status goods," Mazzocco said.
A second study provided more evidence of the role that status plays in conspicuous consumption. In this experiment, 117 white college students were asked to write a story in which they imagined themselves as a character with certain demographic characteristics.
In all cases, the demographic characteristics – including income – remained the same. But half of the students were asked to imagine their character was white, and half were told their character was black.
Afterward, the participants were asked to rate the desirability of high-status and low-status products. Findings showed that the white students who imagined themselves as a black character rated the high-status products as more desirable than did the white students who imagined themselves as white characters. "We called this vicarious conspicuous consumption. White students who temporarily identified with a low-status racial group showed an increased desire for high-status products," Mazzocco said. The findings don't relate only to race, he said. Another study showed that other situations involving status can affect how people feel about conspicuous consumption.
In this experiment, 50 white adults were again asked to write a story imagining themselves as a specific character. In this case, the character was always described as being white. But in half the cases the character was a janitor (a low-status job) and in the other half the character was a brain surgeon (a high-status job).
The findings were clear. Participants who imagined themselves as a janitor had more positive evaluations of high-status products than did the participants who imagined themselves as brain surgeons.

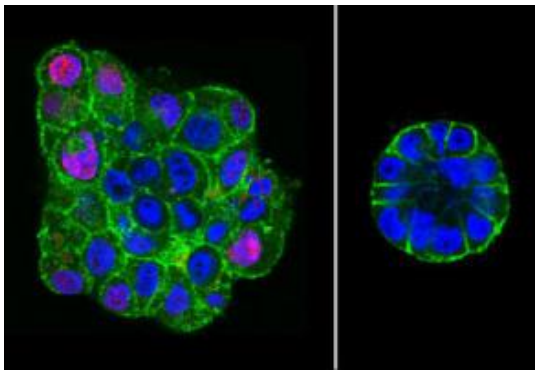
In a final experiment, 69 white adults wrote a story in which they imagined themselves as a white or black character. In this case, they rated their desire to own or purchase specific high- and low-status products. They were then asked to rate the level of social status of the character they wrote about, on a scale of 1 to 10.
In this case, the participants who wrote about the black character were more likely to say they wanted to purchase the high-status products, similar to findings in the earlier studies. And they also rated their character as having lower social status than did the participants who wrote about a white character.
"This provides additional evidence that it is a perception of having low status that is driving the increased preference for high-status products," Mazzocco said. "It suggests that people don't like being in a low-status situation, and they compensate for that by trying to acquire high-status products."
Mazzocco said having this knowledge may help people as they're shopping.
"If you're in a store and find yourself craving an expensive 60-inch flat-screen TV, think about why you want it. It may not be because of the positive attributes of the TV, but because you have a feeling of low status in some part of your life at that time. "Think about parts of your identity where you excel. Maybe you're a good father or mother, or a good student or a good friend. There are many parts to our identity, and it may help to call to mind parts where we feel we have higher status when we're shopping."
Mazzocco said future studies will examine whether people can resist conspicuous consumption when they call to mind parts of their lives where they feel they have high status.

http://www.sciencedaily.com/releases/2012/12/121217140544.htm

To Revert Breast Cancer Cells, Give Them the Squeeze

Researchers at the University of California, Berkeley, and the Lawrence Berkeley National Laboratory have put the squeeze -- literally -- on malignant mammary cells to guide them back into a normal growth pattern.

Dec. 17, 2012 - The findings, presented today (Monday, Dec. 17) at the annual meeting of the American Society for Cell Biology in San Francisco, show for the first time that mechanical forces alone can revert and stop the out-of-control growth of cancer cells. This change happens even though the genetic mutations responsible for malignancy remain, setting up a nature-versus-nurture battle in determining a cell's fate.



"We are showing that tissue organization is sensitive to mechanical inputs from the environment at the beginning stages of growth and development," said principal investigator Daniel Fletcher, professor of bioengineering at UC Berkeley and faculty scientist at the Berkeley Lab. "An early signal, in the form of compression, appears to get these malignant cells back on the right track."

Shown are fluorescence images of uncompressed (left) and compressed (right) colonies of malignant breast epithelial cells. Compressed colonies are smaller and more organized. **Credit: Images courtesy of Fletcher Lab**

Throughout a woman's life, breast tissue grows, shrinks and shifts in a highly organized way in response to changes in her reproductive cycle. For instance, when forming acini, the berry-shaped structures that secrete milk during lactation, healthy breast cells will rotate as they form an organized structure. And, importantly, the cells stop growing when they are supposed to. One of the early hallmarks of breast cancer is the breakdown of this normal growth pattern. Not only do cancer cells continue to grow irregularly when they shouldn't, recent studies have shown that they do not rotate coherently when forming acini.
While the traditional view of cancer development focuses on the genetic mutations within the cell, Mina Bissell, Distinguished Scientist at the Berkeley Lab, conducted pioneering experiments that showed that a malignant cell is not doomed to become a tumor, but that its fate is dependent on its interaction with the surrounding microenvironment. Her experiments showed that manipulation of this environment, through the introduction of biochemical inhibitors, could tame mutated mammary cells into behaving normally.
The latest work from Fletcher's lab, in collaboration with Bissell's lab, takes a major step forward by introducing the concept of mechanical rather than chemical influences on cancer cell growth. Gautham Venugopalan, a member of Fletcher's lab, conducted the new experiments as part of his recently completed Ph.D. dissertation at UC Berkeley.
"People have known for centuries that physical force can influence our bodies," said Venugopalan. "When we lift weights, our muscles get bigger. The force of gravity is essential to keeping our bones strong. Here we show that physical force can play a role in the growth -- and reversion -- of cancer cells."
Venugopalan and collaborators grew malignant breast epithelial cells in a gelatin-like substance that had been injected into flexible silicone chambers. The flexible chambers allowed the researchers to apply a compressive force in the first stages of cell development.

Over time, the compressed malignant cells grew into more organized, healthy-looking acini that resembled normal structures, compared with malignant cells that were not compressed. The researchers used time-lapse microscopy over several days to show that early compression also induced coherent rotation in the malignant cells, a characteristic feature of normal development.
Notably, those cells stopped growing once the breast tissue structure was formed, even though the compressive force had been removed. "Malignant cells have not completely forgotten how to be healthy; they just need the right cues to guide them back into a healthy growth pattern," said Venugopalan.
Researchers further added a drug that blocked E-cadherin, a protein that helps cells adhere to their neighbors. When they did this, the malignant cells returned to their disorganized, cancerous appearance, negating the effects of compression and demonstrating the importance of cell-to-cell communication in organized structure formation.
It should be noted that the researchers are not proposing the development of compression bras as a treatment for breast cancer. "Compression, in and of itself, is not likely to be a therapy," said Fletcher. "But this does give us new clues to track down the molecules and structures that could eventually be targeted for therapies."
The National Institutes of Health helped fund this research through its Physical Science-Oncology program.

http://www.sciencedaily.com/releases/2012/12/121217170950.htm

Hypertension Traced to Source in Brain, Triggering New Paradigm for Hypertension Treatment

When the heart works too hard, the brain may be to blame

When the heart works too hard, the brain may be to blame, says new Cornell University research that is changing how scientists look at high blood pressure (hypertension). The study, published in the Journal of Clinical Investigation in November, traces hypertension to a newfound cellular source in the brain and shows that treatments targeting this area can reverse the disease.
In what peer reviewers are calling "a new paradigm" for tackling the worldwide hypertension epidemic, this insight into its roots could give hope to the billion people it currently afflicts. Hypertension occurs when the force of blood against vessel walls grows strong enough to potentially cause such problems as heart attack, stroke and heart or kidney disease. The heart pumps harder, and often the hormone angiotensin-II (AngII) gets the pressure cooking by triggering nerve cells that constrict blood vessels.
"We knew the central nervous system orchestrates this process, and now we've found the conductor," said Robin Davisson, a professor of molecular physiology with a joint appointment at Cornell's College of Veterinary Medicine and Weill Cornell Medical College.
Two-thirds of Americans have hypertension, which is the leading cause of North America's No. 1 killer: heart disease, according to the Centers for Disease Control and Prevention.
Davisson's lab traced neurochemical signals back to endoplasmic reticulum (ER), the protein factory and stress-management control center in every cell. If something goes wrong in a cell, the ER activates processes to adapt to the stress. Long-term ER stress can cause chronic disease, and several stressors that ER responds to have been connected to hypertension. Davisson's lab found that high levels of AngII put stress on the ER, which responds by triggering the cascade of neural and hormonal signals that start hypertension.
But not just any cell's ER can conduct this complex orchestra. Those that can trigger the signal cascade are clustered near the bottom of the brain in a gate-like structure called the subfornical organ (SFO). Unlike most of the brain, the SFO hangs outside a protective barrier that keeps most circulating particles from entering the brain. The SFO can interact with particles like AngII that are too big to cross through and can also communicate with the brain's inner chambers.
This is good news for developing therapies -- because the SFO sits outside the barrier, it can be reached through such normal treatment routes as pills or injections rather than riskier brain procedures. Davisson's lab showed that treatments that inhibit ER stress in the SFO can completely stop AngII-based hypertension and lower blood pressure to normal levels.
"Our work provides the first evidence that higher levels of AngII cause ER stress in the SFO, that this causes hypertension, and that we can do something about it," said Davisson. "This finding may also suggest a role for ER stress in hypertension types that don't involve AngII, like some spontaneous or genetic forms."
Inspired by the paradigm shift that this study has sparked, the editors of the Journal of Clinical Investigation published a commentary concluding that this discovery "opens new avenues for investigation and may lead to new therapeutic approaches for this disease."
Colin N. Young, Xian Cao, Mallikarjuna R. Guruju, Joseph P. Pierce, Donald A. Morgan, Gang Wang, Costantino Iadecola, Allyn L. Mark, Robin L. Davisson. ER stress in the brain subfornical organ mediates angiotensin-dependent hypertension. Journal of Clinical Investigation, 2012; 122 (11): 3960 DOI: 10.1172/JCI64583

http://www.eurekalert.org/pub_releases/2012-12/plos-let121712.php

Little evidence to support TB interventions in real-world, low-resource settings

Little evidence from real world situations in low-and-middle income countries to support effectiveness and financial value of TB interventions recommended by the World Health Organization

There is little evidence from real world situations in low-and-middle income countries to support the effectiveness and financial value of five interventions* recommended by the World Health Organization (WHO) to control tuberculosis, which may be a reason why these interventions have not been implemented in many countries, according to a study by international experts published in this week's PLOS Medicine.

Over the past few years, WHO has recommended that countries implement several interventions to help control the spread of tuberculosis through measures to improve prevention, diagnosis, and treatment. The authors led by Frank Cobelens from the Amsterdam Institute of Global Health and Development in the Netherlands reviewed 208 appropriate studies to investigate the evidence for the effectiveness of each of these interventions and also for additional information on the setting and conditions of implemented interventions, which might be useful to other countries.

The authors found that there were very few real-world studies reporting on the effectiveness of interventions in program settings (rather than in optimal conditions under research settings). Few studies evaluated the methods used to implement the intervention or addressed delivery and operational issues (such as adherence to treatment) and there were few economic evaluations of the recommended interventions.

The authors say: "There are substantial gaps in published evidence for scale-up for five WHO-recommended TB interventions settings at country level, which for many countries possibly precludes program-wide implementation of these interventions." They continue: "This lack of "evidence for scale-up" may be an important cause of the shortfall in implementation of these interventions in many countries."

The authors add: "There is a strong need for rigorous operational research studies to be carried out in programmatic settings to inform on best of existing and new interventions in TB control."

Five key interventions *currently recommended by WHO guidelines are: treatment with isoniazid to prevent TB among people who are HIV positive, and also among household contacts of people infected with TB; the use of clinical pathways (algorithms) for diagnosing TB in people accessing health care who have a negative smear test - the most commonly used diagnostic test, which relies on sputum samples - ("rule-in algorithms"); screening algorithms for excluding TB in people who have HIV ("rule-out algorithms"); and finally, provision of second-line treatment for multidrug-resistant tuberculosis (a form of TB that does not respond to the most commonly used drugs).*

Funding: *The Stop TB Partnership and the Global Fund to fight AIDS, Tuberculosis and Malaria (GFATM) were involved in the study design and the preparation of the manuscript as this study was commissioned within a larger frame project aiming at identifying priorities for operational research in tuberculosis, with the view to assist countries in preparing and conducting operational research to improve tuberculosis control. Results of the study were presented and discussed in a workshop laying the ground for a manual on TB operational research. The Stop TB Partnership and GFATM had no influence on the analysis, data interpretation and results, or in the decision to submit the (revised) manuscript for publication.*

Competing Interests: The authors have declared that no competing interests exist.

Citation: *Cobelens F, van Kampen S, Ochodo E, Atun R, Lienhardt C (2012) Research on Implementation of Interventions in Tuberculosis Control in Low- and Middle-Income Countries: A Systematic Review. PLoS Med 9(12): e1001358. doi:10.1371/journal.pmed.1001358*

http://phys.org/news/2012-12-armour-pompeii-japan.html

Remains of man in armour found at 'Pompeii of Japan'

The remains of a high-caste man wearing armour who was buried by hot ash - possibly as he tried to calm the wrath of an erupting volcano - have been found in an area known as the "Pompeii of Japan".

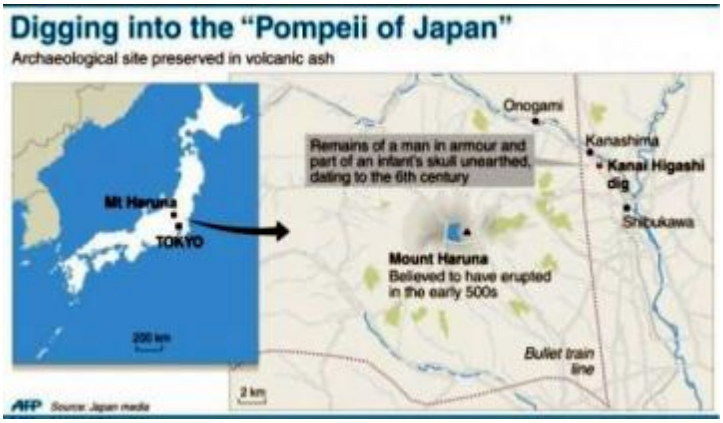
Archaeologists say they have unearthed the well-preserved body of a sixth-century man who had apparently turned to face a flow of molten rock as it gushed through his settlement.

"Under normal circumstances, you would flee if pyroclastic flows are rushing toward you and bringing waves of heat. But this person died facing it," said Shinichiro Ohki, of Gunma Archaeological Research Foundation.

"Maybe, if he were someone of a high position, he might have been praying, or doing something in the direction of the volcano and attempting to appease its anger," Ohki told AFP on Monday.

This handout picture taken by Gunma Archaeological Research Foundation on November 30, 2012 shows a well-preserved body of a sixth-century man in a suit of armour (yellow), found at the Kanai Higashiura dig in Gunma prefecture, 110km north of Tokyo. The man had apparently turned to face a flow of molten rock as it gushed through his settlement.

The remains, along with a part of an infant's skull, were found in the Kanai Higashiura dig in Gunma prefecture, roughly 110 kilometres (70 miles) northwest of Tokyo, at the site of the volcanic Mount Haruna.



The find comes from an area known to enthusiasts as the "Pompeii of Japan" a reference to the Roman city near modern-day Naples buried by the eruption of Mount Vesuvius in AD79.

The body is clad in a relatively sophisticated kind of armour made by craftsmen who bound small iron plates with thin leather strips, which would have represented the latest technological import from the Korean Peninsula.

It may have been brought to Japan after the practice of horse riding was introduced in the late fifth century, Ohki said, adding that the armour was much more sophisticated than the single-plate type common in the period.

"It indicates the person wearing it was someone of a high position, like a regional leader," Ohki told AFP, adding studies would be carried out to see if the man was related to occupants of ancient tombs dotting the region.

Archaeologists will also examine the bones to determine whether the man and the child were related.

"If possible, we would like to study their DNA. Were they related? Why and how did they die there?" Ohki said.

http://www.bbc.co.uk/news/uk-england-essex-20762437

Cystic fibrosis woman died with smoker's donor lungs

A 27-year-old woman with cystic fibrosis died of cancer after she was given the donor lungs of a smoker.

Jennifer Wederell, of Hawkwell, Essex, died at home in August - 16 months after the transplant at Harefield Hospital in London.

Colin Grannell said he believes his daughter would not have agreed to the transplant had she known the middle-aged donor was a heavy smoker.

The hospital has apologised for not giving her that choice.

Jennifer had been diagnosed with cystic fibrosis at the age of two and by her mid-20s was using oxygen 24 hours a day.

She had been on the waiting list for a lung transplant for 18 months when in April 2011, she was told there had been a match.

Mr Grannell said the family had "lived all for that moment" for years, and thought it would help Jennifer "cheat" her condition.

She married her fiance David Wederell in September last year, but by February 2012 a malignant mass was found in her lungs.

'Should have choice'

"The shock immediately turned to anger in so far as all the risks were explained in the hour before her transplant and not once was the fact smoker's lungs would be used mentioned," said Mr Grannell.

"She was dying a death that was meant for someone else."

He has set up a Facebook group, Jennifer's Choice, to encourage non-smokers to sign up to the organ donor register.

The Royal Brompton and Harefield NHS Foundation Trust said: "It is very rare for patients to specify that they do not wish to be considered for clinically healthy lungs from smokers.

"This is because the risks are much higher if patients decline donor lungs from a former smoker, and decide to wait for another set of organs which are both a match for them and from a non-smoker, to become available.

"However, we recognise that Jennifer should have been given the opportunity to make this choice.

"We have apologised sincerely for this oversight. "Regrettably, the number of lungs available for transplantation would fall by 40% if there was a policy of refusing those which have come from a smoker; waiting lists would increase and many more patients would die without a transplant."

Prof Stephen Spiro, honorary medical adviser at the British Lung Foundation said: "It's very difficult to say why the cancer developed.

"It is seriously possible that the cancer may have started in the lung before transplantation, but it was so small there was no chance of spotting it. "Recipients of transplants are immunosuppressed, to stop the body rejecting the organ - this may have encouraged the cancer to grow. But is no one really knows- these are just theories."

http://www.bbc.co.uk/news/health-20758754

Norovirus: The winter vomiting bug that is hard to conquer

Norovirus is the Ferrari of the virus world," says Professor Ian Goodfellow, a Wellcome Trust fellow

By Smitha Mundasad BBC News

'Vomiting Larry' is busy being sick over and over again in an experiment to test just how far the winter vomiting bug can travel when it makes you ill.Lucky for Larry, he is not a constantly retching human - but a simulated vomiting system that shows the virus can travel an impressive 3m (9.8ft) in a projectile episode, according to his creators at the Health and Safety Laboratory.

The winter vomiting bug (norovirus) has been responsible for an estimated 880,000 cases of vomiting and diarrhoea in the UK since the summer. It is a hardy virus that clearly spreads with ease - one of the few infections you really can catch from a toilet seat, or even from the air in the bathroom if an infected person has recently pulled the flush. Luckily most people make a full recovery in a few days, but for anyone who is already vulnerable - people who are already unwell or in their later years for example - it can pose a serious threat.

It was first noticed when 150 children at the Norwalk-Bronson Elementary School in Ohio were all struck down with the bug in 1968.

This 40-year-old incident is now eternalised in medical history, making up the first part of the virus's name.

And unsuspecting groups of students continue to be knocked down by its tenacious grip on the small intestine.

A girls' football team from Canada was taken ill after a team-mate developed the illness - but she had had no contact with them. The culprit was a grocery bag in the corner of the bathroom she had used.

Computer model of the virus One in 20 people in the UK suffer from norovirus each year

Aerosolised particles of the virus landed on the grocery bag, and spread to members of the team who touched the bag or ate the packaged crisps and cookies inside it, according to research published in the Journal of **Infectious Diseases.**

And it can stick around for a long time.

It is widely accepted that it can last for two weeks on hard surfaces, says Professor Ian Goodfellow of the University of Cambridge.

It is not only its ability to travel so far or survive on everyday surfaces that makes it, in viral terms, "the ideal infectious agent", says Dr Aron Hall, from the Centers for Disease Control and Prevention in the US.

'Rapid reproduction'

As few as 18 viral particles can infect a new person, but there are often thousands of particles present in each drop of vomit. And it is able to reproduce at a rapid speed - thousands of times more quickly than humans.

NOROVIRUS

Symptoms include vomiting and/or diarrhoea

You may also have a fever, headache and stomach cramps

Over-the-counter medicines can be useful in treating headaches and other aches and pains

Do not visit your GP surgery or A&E unit if you have it

If symptoms persist for more than three or four days, or if you have a serious illness, seek medical attention through contact with your GP

Wash hands thoroughly, particularly after using the toilet and before eating

Clean hard surfaces with detergent followed by disinfection with a bleach solution, paying particular attention to the toilet and surrounding area

Source: Health Protection Agency

The fact it makes most of us only mildly or moderately ill is all part of its high-achieving strategy. If it made people more seriously ill, or even killed them, it would not be able to spread so well. "From an evolutionary stand point if you kill your host you are not going to have anywhere to live, so keeping your host alive has its advantages," says Dr Hall.

All these factors make it very difficult to stop in its tracks. That is, apart from the good old fashioned method of washing your hands with soap and water.

According to a study of seven outbreaks at an international scout jamboree, each boy scout who had the infection passed it on to 14 others until enhanced hygiene measures were imposed. This included separate bathrooms for infected people and strict hand-washing regimes. The number of infections passed on per person then went down to two - but still not enough to stop it from spreading completely.

The virus also manages to evade many commonly used alcohol gels and some domestic cleaning products.

You are safe with thoroughly cooked food, but it can escape freezing and mild heating.

Scientists have spent many years trying to find a way to stop it from closing hospital wards, leaving offices short-staffed in winter months and causing chaos on cruise ships. Legend has it that the first of the now many attempts to study the virus involved volunteers who drank the filtered diarrhoea of people who had the infection. Study designs may have moved on a lot since then, but we are still far from finding a cure.

One of the reasons is that no one has been able to persuade the virus to grow in a lab, says Prof Ian Goodfellow:

"In my lab we are trying to understand how these viruses work, with the overall aim of trying to identify a drug that will prevent infection and control outbreaks when they do occur," he says.
But no one has yet pinned down why it is so elusive. And until a vaccine or cure is found it is likely to continue to put some people off their turkey this Christmas.

http://www.sciencedaily.com/releases/2012/12/121218111921.htm

Silent Stroke Can Cause Parkinson's Disease

Scientists at The University of Manchester have for the first time identified why a patient who appears outwardly healthy may develop Parkinson's disease.

Whilst conditions such as a severe stroke have been linked to the disease, for many sufferers the tremors and other symptoms of Parkinson's disease can appear to come out of the blue. Researchers at the university's Faculty of Life Sciences have now discovered that a small stroke, also known as a silent stroke, can cause Parkinson's disease. Their findings have been published in the journal Brain, Behavior, and Immunity.
Unlike a severe stroke, a silent stroke can show no outward symptoms of having taken place. It happens when a blood vessel in the brain is blocked for only a very short amount of time and often a patient won't know they have suffered from one. However, it now appears one of the lasting effects of a silent stroke can be the death of dopaminergic neurons in the substantia nigra in the brain, which is an important region for movement coordination.
Dr. Emmanuel Pinteaux led the research: "At the moment we don't know why dopaminergic neurons start to die in the brain and therefore why people get Parkinson's disease. There have been suggestions that oxidative stress and aging are responsible. What we wanted to do in our study was to look at what happens in the brain away from the immediate area where a silent stroke has occurred and whether that could lead to damage that might result in Parkinson's disease."
The team induced a mild stroke similar to a silent stroke in the striatum area of the brain in mice. They found there was inflammation and brain damage in the striatum following the stroke, which they had expected. What the researchers didn't expect was the impact on another area of the brain, the substantia nigra. When they analysed the substantia nigra they recorded a rapid loss of Substance P (a key chemical involved in its functions) as well as inflammation. The team then analysed changes in the brain six days after the mild stroke and found neurodegeneration in the substantia nigra. Dopaminergic neurons had been killed.
Talking about the findings Dr Pinteaux said: "It is well known that inflammation following a stroke can be very damaging to the brain. But what we didn't fully appreciate was the impact on areas of the brain away from the location of the stroke. Our work identifying that a silent stroke can lead to Parkinson's disease shows it is more important than ever to ensure stroke patients have swift access to anti-inflammatory medication. These drugs could potentially either delay or stop the on-set of Parkinson's disease."
Dr Pinteaux continued: "What our findings also point to is the importance of maintaining a healthy lifestyle. There are already guidelines about exercise and healthy eating to help reduce the risk of having a stroke and our research suggests that a healthy lifestyle can be applied to Parkinson's disease as well."
Following the publication of these findings, Dr Pinteaux hopes to set up a clinical investigation on people who have had a silent stroke to assess dopaminergic neuron degeneration. In the meantime he will be working closely will colleagues at The University of Manchester to better understand the mechanisms of inflammation in the substantia nigra.
Beatriz Rodriguez-Grande, Victoria Blackabey, Beatrice Gittens, Emmanuel Pinteaux, Adam Denes. Loss of Substance P and inflammation precede delayed neurodegeneration in the substantia nigra after cerebral ischemia. Brain, Behavior, and Immunity, 2012; DOI: 10.1016/j.bbi.2012.11.017

http://www.sciencedaily.com/releases/2012/12/121218121216.htm

Psychologists: Scrooge's Transformation Parallels Real Life-Changing Experiences

"Bah, humbug!" is the line most closely associated with Ebenezer Scrooge, the famous miser from "A Christmas Carol." But the authors of a new study on life-changing experiences give author Charles Dickens high marks for his portrayal of Scrooge's sudden switch to saintliness.

Former grad student Jon Skalski and Brigham Young University psychology professor Sam Hardy conducted an in-depth study of 14 people who experienced profound, sudden and lasting change. They say the fictional Scrooge would fit right in.
"Like our participants, Scrooge was suffering," Skalski said. "There was disintegration. There was a world that was ripe for change because of suffering going on."
Though Scrooge had money, he hit rock bottom in terms of relationships. Orphaned as a child and broken-hearted from a failed engagement, Scrooge's pains intensify each Christmas Eve, the anniversary of the death of his only friend, Jacob Marley.

In the story, Marley appears seven years after his death as a voice of warning. Though a ghost, the role he plays is true to life. Most study participants described the presence of a trusted other person during their experience. "Just by their presence, a trusted friend can open up possibilities and a sense of faith in what's possible that one can't see," Skalski said.
Skalski and Hardy's research will appear in the January issue of The Humanistic Psychologist. Finding people that fit the criteria was no easy task. To do so, they employed ads on Craigslist in Illinois and Utah.
Notably, the experiences shared by the participants were not recent events. On average, nine years had passed between the transformation and their interview. Most of them could remember the exact time of day when the turning point occurred.
"I've often thought about this, whether these transformations are really sudden or gradual," Skalski said. "It's like water boiling -- you can look at that as a discontinuous change from not boiling to boiling, but there are certain elements going on beneath the surface that allow for the dramatic change to take place."
For an entrepreneur referred to as Kevin in the study, the preceding turmoil arose because his identity as a successful businessman crashed along with his failed ventures. Like Scrooge, he had neglected relationships and said his psyche was "in a very dark place."
But with his breakthrough moment, life instantly took on a whole new meaning for Kevin.
"I say it's the best thing that could've happened, because my life is so much more rewarding than it once was. You can't put a price tag on certain…events that I maybe missed before -- certain events, and a marriage, and a family, birthdays, you know? Certain things that are just really fun to be a part of are more meaningful, and it is happiness -- the kind that lasts. I know these truths have been around forever. But for me they're new."
Similarly, another participant's world crumbled because she based her worth on how well she did in school. Like Scrooge and Kevin, she emerged with a focus on other people.
"Now I measure success by my -- how much time I spend serving and doing those things, because those -- serving and being with people -- are really what bring me satisfaction now."
Each of the study participants experienced overwhelming stress prior to their breakthrough. Hardy, an expert in human development, wonders whether hitting rock bottom is a necessary ingredient for such positive transformations.
"That led me to think, well, is there a way that people can capitalize on these mechanisms of change and initiate them themselves instead of bottoming out," Hardy said. "Can you self-initiate this kind of change?"
Skalski sees another holiday parallel with his research in the film "It's a Wonderful Life." After planning to end his life, George Bailey realizes how other people depend on him in Bedford Falls, prompting his famous line, "I want to live again!"
"Those stories are stuck within our culture," Skalski said. "We all know deep down inside that human beings can and do change in profound and significant ways."
Skalski is seeking a Ph.D. in psychology at the University of West Georgia.

http://www.bbc.co.uk/news/health-20770922

Men's cancer risk 'to climb to one in two'

Men look set to have a one in two chance of developing cancer in their lifetime, UK experts predict.

By Michelle Roberts Health editor, BBC News online

The increase to 50 out of 100, up from the current 44 in 100 chance, is largely down to people living longer - age is the biggest cancer risk factor, says Cancer Research UK. The cancers set to increase the most in men within the next 15 years are bowel, prostate and skin (melanoma). But more will survive cancer, thanks to better screening and treatments. Medical advances mean cancer survival has already doubled in the past 40 years. And with more research, experts believe outcomes could continue to improve.

Projections

The team from the Wolfson Institute of Preventative Medicine at Queen Mary, University of London, and Cancer Research UK reached their figures by looking at past cancer incidence and mortality rates and projected population data for the UK. They predict that by 2027 some 416,000 UK people are expected to be diagnosed with cancer compared with about 324,000 diagnosed in 2010. For men, the figure will be more than 221,000, up from 164,000 in 2010. And there will be more than 194,000 women diagnosed with cancer in 2027 compared with 160,000 in 2010 - which would mean a woman's lifetime odds of developing cancer would be 44 in 100, up from 40 in 100.
Dr Harpal Kumar of Cancer Research UK said the figures provided a glimpse into the future and what challenges lie ahead. A pressing task is to find an effective way to screen for prostate cancer.

Not all cancer in the prostate is aggressive or life-threatening - some people live with the condition for a lifetime without any problems.
But doctors still have no reliable test that can spot which of these tumours are safe to leave alone.
Another challenge is getting men to turn up for cancer screening even when a good test for it does exist, says Alan White, chairman of the Men's Health Forum and professor of men's health at Leeds Metropolitan University.
For example, although men tend to be at greater risk of developing bowel cancer than women, relatively fewer men than women go for screening for this cancer, says Prof White. "It's desperately important that men take up any opportunity to go for cancer screening that they can.
"Some men are fatalistic about cancer and screening. But screening does make a difference. If cancers are spotted earlier they are easier to treat. "We also know that men who discuss screening with their doctor or their partner are more likely to take up the offer."
He said it was important for people to realise that there is a lot we can do ourselves to lower our own cancer risk, including limiting how much alcohol you drink, giving up smoking, getting enough exercise and eating a healthy diet. Experts estimate that about four in every 10 cases of cancer could be avoided in this way.
In England, screening is available for bowel, breast and cervical (neck of the womb) cancer.
Men can also request medical tests (a prostate specific antigen blood test) if they are concerned about prostate cancer, although these checks are less than perfect.

http://www.eurekalert.org/pub_releases/2012-12/cfb-sci121912.php

Small changes in eating prompts weight loss

The mindless eating challenge: Evaluation of a public web-based healthy eating and weight loss program

Making small easy changes to our eating habits on a consistent basis - 25 days or more per month - can lead to sustainable weight loss, according to research by Professor Brian Wansink in Cornell University's Food and Brand Lab. The challenge is to figure out which changes work for specific individuals and how to stick with changes long enough to make them second nature.
To explore this issue, Cornell researchers launched the National Mindless Eating Challenge (NMEC), an online healthy eating and weight loss program that focused on simple eating behavior changes, instead of dieting. NMEC participants, after answering questions about their eating goals, background and well-being, were sent three customized tips to follow for a month. All tips were founded on research and based on Wansink's book "Mindless Eating: Why We Eat More than We Think" (Bantam, 2006). Participants could download a checklist to track their adherence to tips and receive email reminders to keep them on track. At the end of each month they were expected to send in a follow-up survey. Of the 504 participants who completed at least one follow-up survey, more than two thirds (42 percent) either lost weight or maintained their weight (27 percent).
Weight loss was highest among people who made changes consistently.
Those whose adherence was 25 or more days per month reported an average monthly weight loss of 2 pounds, and those who stayed in the program at least three months and completed at least two follow-up surveys lost on average 1 percent of their initial weight.
According to the study, which is published in the peer-reviewed Journal of Medical Internet Research (Nov-Dec., Vol. 14:6), common barriers that prevented people from making changes included personally unsuitable tips, forgetting, being too busy, unusual circumstances such as vacations and emotional eating.
"These results confirm that small, consistent changes in our daily eating behavior can result in gradual weight loss and in developing healthier eating habits," said Wansink, a marketing professor in Cornell's Dyson School of Applied Economics and Management. Results of the study also show that it is a challenge for many people to stick to a program for a long period of time. For those who want to lose weight or eat more healthfully, the researchers conclude that finding an initial set of tips that are relevant and doable for an individual can be enough to learn the general principle.
"Later come up with your own changes and succeed at reaching your goal," Wansink said.
The NMEC participants said that the most effective tips they received were:

Keep counters clear of all foods but the healthy ones.
Never eat directly from a package – always portion food out onto a dish.
Eat something hot for breakfast within the first hour of waking up.
Avoid going more than thee or four hours without having something small to eat.
Put down your utensils between bites to slow down your eating.

http://www.eurekalert.org/pub_releases/2012-12/haog-adc121912.php

Alzheimer's disease: Cutting off immune response promises new approach to therapy

In the case of Alzheimer's disease, the activation of the molecular alarm may have negative consequences

The Bonn site of the German Center for Neurodegenerative Diseases (DZNE) and the University of Bonn are leading contributors.

The complex named "NLRP3 inflammasome" is composed of several proteins and plays a key role in the immune system. It resembles a fire alarm sensor that triggers a chain reaction when activated. As a result, immune cells are mobilized and substances that foster inflammation are released. This process can be triggered by infections, which are subsequently suppressed by the immune response. However, in the case of Alzheimer's disease, the activation of the molecular alarm may have negative consequences: nerve cells are damaged and die. Ultimately, this leads to the loss of brain function and mental capabilities in humans.

Alzheimer's disease is accompanied with deposits in the brain. That these so-called "plaques" have the capability to activate the NLRP3 inflammasome had already been identified by investigating individual cells. But the exact effect on the organism was unknown. "It was unclear what consequences an increased activity of the NLRP3 inflammasome could have on the brain," explains Prof. Michael Heneka, who conducts research at both, the DZNE and the University of Bonn. Working in a team with immune researcher Eicke Latz as well as with other colleagues, Heneka has now been able to show that the protein complex does in fact play a determining role in the development of Alzheimer's disease.

Studies involved humans and mice

The researchers collected a comprehensive chain of evidences: they examined both the brains of deceased Alzheimer patients and of mice who exhibited behavioural disorders that are typically associated with Alzheimer's disease. The researchers found an activated form of the NLRP3 inflammasome in both cases. Looking at another group of mice, the scientists examined possibilities for suppressing inflammatory reactions. To achieve this, they removed the genes that trigger production of the NLRP3 inflammasome. Therefore, these mice were no longer able to synthesize the protein complex. As a result, the animals developed only relatively mild symptoms of the disease. Moreover, their brains showed only reduced amounts of the damaging plaques.

"We have stumbled upon a critical factor in the development process of Alzheimer's. Given these findings it appears to be a very promising possibility to block the activity of the inflammasome," comments Heneka. In his view, proper pharmaceuticals might be able to stop a chain reaction that would otherwise result in the inflammation of brain cells. "At present various options are being pursued to act upon the course of the disease," says the neuroscientist. "Our results points to a new possibility. Nevertheless, we are still in the process of doing basic research."

However, the group of scientists in Bonn is already making plans for the future. Eicke Latz's team, which also made significant contributions to the latest study, has already begun to search for active components that could block the NLRP3 inflammasome. "The testing of potential substances in the laboratory would be a next step. We hope to start as early as next year," says Heneka.

***"NLRP3 is activated in Alzheimer´s disease and contributes to pathology in APP/PS1 mice**," Michael T. Heneka, Markus P. Kummer, Andrea Stutz, Andrea Delekate, Stephanie Schwartz, Ana Vieira Saecker, Angelika Griep, Daisy Axt, Anita Remus, Te-Chen Tzeng, Ellen Gelpi, Annett Halle, Martin Korte, Eicke Latz, Douglas Golenbock, Nature, DOI: 10.1038/nature11729*

http://www.eurekalert.org/pub_releases/2012-12/jhm-jhm121912.php

Johns Hopkins malpractice study: Surgical 'never events' occur at least 4,000 times per year

Researchers advocate public reporting of mistakes

After a cautious and rigorous analysis of national malpractice claims, Johns Hopkins patient safety researchers estimate that a surgeon in the United States leaves a foreign object such as a sponge or a towel inside a patient's body after an operation 39 times a week, performs the wrong procedure on a patient 20 times a week and operates on the wrong body site 20 times a week. The researchers, reporting online in the journal Surgery, say they estimate that 80,000 of these so-called "never events" occurred in American hospitals between 1990 and 2010 - and believe their estimates are likely on the low side.

The findings - the first of their kind, it is believed - quantify the national rate of "never events," occurrences for which there is universal professional agreement that they should never happen during surgery. Documenting the magnitude of the problem, the researchers say, is an important step in developing better systems to ensure never events live up to their name.

"There are mistakes in health care that are not preventable. Infection rates will likely never get down to zero even if everyone does everything right, for example," says study leader Marty Makary, M.D., M.P.H., an

associate professor of surgery at the Johns Hopkins University School of Medicine. "But the events we've estimated are totally preventable. This study highlights that we are nowhere near where we should be and there's a lot of work to be done."

For the study, Makary and his colleagues used the National Practitioner Data Bank (NPDB), a federal repository of medical malpractice claims, to identify malpractice judgments and out-of-court settlements related to retained-foreign-body (leaving a sponge or other object inside a patient), wrong-site, wrong-procedure and wrong-patient surgeries. They identified 9,744 paid malpractice judgments and claims over those 20 years, with payments totaling $1.3 billion. Death occurred in 6.6 percent of patients, permanent injury in 32.9 percent and temporary injury in 59.2 percent.

Using published rates of surgical adverse events resulting in a malpractice claim, the researchers estimate that 4,044 surgical never events occur in the United States each year. The more serious the outcome, the more the patient (or his family) was paid.

Makary says the NPDB is the best source of information about malpractice claims for never events because these are not the sort of claims for which frivolous lawsuits are filed or settlements made to avoid jury trials. "There's good reason to believe these were all legitimate claims," he says. "A claim of a sponge left behind, for example, can be proven by taking an X-ray."

By law, hospitals are required to report never events that result in a settlement or judgment to the NPDB. If anything, he says, his team's estimates of never events are low because not all items left behind after surgery are discovered. Typically, they are found only when a patient experiences a complication after surgery and efforts are made to find out why, Makary says.

In their study, never events occurred most often among patients between the ages of 40 and 49, and surgeons in this same age group were responsible for more than one-third of the events, compared to 14.4 percent for surgeons over the age of 60. Sixty-two percent of the surgeons were cited in more than one separate malpractice report, and 12.4 percent were named in separate surgical never events.

Makary notes that at many medical centers, patient safety procedures have long been in place to prevent never events, including mandatory "timeouts" in the operating room before operations begin to make sure medical records and surgical plans match the patient on the table. Other steps include using indelible ink to mark the site of the surgery before the patient goes under anesthesia. Procedures have long been in place to count sponges, towels and other surgical items before and after surgery, but these efforts are not foolproof, Makary notes.

Many hospitals are moving toward electronic bar codes on instruments and materials to enable precise counts and prevent human error. Surgical checklists, pioneered at The Johns Hopkins Hospital, are also often in place.

Along with better procedures to prevent never events, better reporting systems are needed to speed up safety efforts, says Makary. He advocates public reporting of never events, an action that would give consumers the information to make more informed choices about where to undergo surgery, as well as "put hospitals under the gun to make things safer."

Currently, he notes, hospitals are supposed to voluntarily share never event information with the Joint Commission that assesses hospital safety and practice standards, but that doesn't always happen.

Other Johns Hopkins researchers who contributed to the study include Winta T. Mehtsun, M.D., M.P.H.; Andrew M. Ibrahim, M.D.; Marie Diener-West, Ph.D.; and Peter J. Pronovost, M.D., Ph.D.

http://www.eurekalert.org/pub_releases/2012-12/ci-css121812.php

Closest sun-like star may have planets

An international team of scientists, including Carnegie's Paul Butler, has discovered that Tau Ceti, one of the closest and most Sun-like stars, may have five planets. Their work is published by Astronomy & Astrophysics and is available online.

Washington, D.C.- At a distance of twelve light years and visible with a naked eye in the evening sky, Tau Ceti is the closest single star with the same spectral classification as our Sun. Its five planets are estimated to have masses between two and six times the mass of the Earth--making it the lowest-mass planetary system yet detected. One of the planets lies in the habitable zone of the star and has a mass around five times that of Earth, making it the smallest planet found to be orbiting in the habitable zone of a Sun-like star.

The international team of astronomers, led by Mikko Tuomi from the University of Hertfordshire, combined more than six-thousand observations from three different instruments and applied intensive modeling to the data. Using new techniques, the team found a method to find signals half the size previously thought possible, which greatly improves the sensitivity of searches for small planets and suggests that Tau Ceti is not a lone star but has a planetary system.

"We pioneered new data modeling techniques by adding artificial signals to the data and testing our recovery of the signals with a variety of different approaches," Tuomi said. "This significantly improved our noise modeling techniques and increased our sensitivity to find low mass planets."
Tau Ceti was chosen for this noise-modeling study because the team thought it had no signals and would be a good benchmark system to test their methods for planet detection. This is particularly true because it is so bright and similar to our own Sun. It's also one of Earth's nearest cosmic neighbors, so scientists could be able to learn about the atmospheres of these planets in the not-too-distant future.
More than 800 planets have been discovered orbiting other worlds, but planets in orbit the around the nearest Sun-like stars are particularly valuable for research.
"We are now glimpsing for the first time the secrets of our nearest companion stars and their previously hidden reservoirs of potentially habitable planets," Butler said. "This work presages the time when we will be able to directly see these planets, and search them for water, carbon dioxide, methane, and other signposts of life."
The work herein is based in part on observations obtained at the W. M.Keck Observatory, which is operated jointly by the University of California and the California Institute of Technology.
This research is supported by Planets Around Cool Stars, a Marie Curie Initial Training Network funded by the European Commission's Seventh Framework Programme. Fondecyt, Centro de Astrofísica, the GEMINI-CONICYT FUND, the Comit´e Mixto ESO-GOBIERNO DE CHILE, the NSF, the Australian government.

http://news.discovery.com/earth/plants-smell-fruit-flies-funk-121219.html#mkcpgn=rssnws1

Plants Smell Fruit Flies' Funk

The flowers we smell may be getting a whiff of us at the same time.

Analysis by Tim Wall

No one knows if roses take time in life to stop and smell the humans, but some plants take action when they smell insect pests.
A study at Penn State found that when tall goldenrod plants sense the sex attractant released by male fruit flies, they produce their own chemical defenses. Those defenses make the plants less appealing to female fruit flies looking for a place to lay their eggs. Females puncture the plants and lay their eggs inside the stems. The attack isn't deadly, but plants serving as fruit fly nurseries tend to produce fewer and smaller seeds.
However, when goldenrod plants in the wild had been exposed to male fruit fly's amorous odor, the plants tended to harbor fewer egg-laying sites. What's more the plants also became more resistant to attacks by other insects.
The exact physiological means by which the plants smell the flies is still a mystery. "Our understanding of plant olfaction in general remains quite limited," said Mark Mescher, an entomologist at Penn State, in a press release.
"It's become increasingly clear in recent years that plants are responsive to odors," said Mescher. "But previous examples of this are all plant-to-plant. For example, some plants have been shown to respond to the odor of insect-damaged neighbors by priming their own defenses. What's new about this is that it seems that plants may sometimes be able to smell the insects themselves."
The study was published in the Proceedings of the National Academy of Sciences.

http://phys.org/news/2012-12-growth-survival-superorganism-ant-colonies.html

Research predicts growth, survival of 'superorganism' ant colonies

Research that views ant colonies as "superorganisms" in which social insects function much like the cells of a body

Phys.org - Smaller ant colonies tend to live faster, die younger and burn up more energy than their larger counterparts, as do the individual ants that make up those colonies, according to new research that views the colonies as "superorganisms" in which social insects function much like the cells of a body.
The research, published in the Dec. 23 issue of the journal Biology Letters, describes a new mathematical model that can predict the survival, growth and life span of ant colonies. Dr. Chen Hou, an assistant professor of biological sciences at Missouri University of Science and Technology, is one of the authors of the study and developed the mathematical models used to predict colony growth and survival.
Hou also collected some of the data used for the Biology Letters study, titled "Towards a general life-history model of the superorganism: predicting the survival, growth and reproduction of ant societies."
In the article, Hou and his colleagues compared the rates of metabolism, growth, reproduction and longevity of individual ants with those same traits for entire colonies. He based his models on Kleiber's Law, the observation that the metabolic rate for organisms - the rate at which they process and use energy over time - tends to increase at a rate that is to the 3/4 power of that organism's body mass. Named after Max Kleiber's biological work in the early 1930s, Kleiber's Law is also known as "quarter-power scaling."

As Hou explains it, a horse may be 10,000 times heavier than a mouse, but it doesn't consume 10,000 times more energy. Applying quarter-power scaling, researchers can determine that a horse, which is 10,000 times larger than the mouse, only consumes 1,000 times more energy (because 10,000 to the 3/4 power equals 1,000).
The same phenomena holds true at the cellular level, Hou says. Two similar organ cells from two different organisms -a mouse and a horse, for instance - do not use proportionately equal amounts of energy. "The one from the horse needs and uses much less energy than the one from the mouse," even though both cells have the same purpose and function, Hou says.
In ant colonies, that same phenomenon applies, Hou and his colleagues point out in the Biology Letters study. The ants behave more like the cells of animal and their colonies more like the animal itself - which is why the researchers classify colonies as "superorganisms." The individual ants of larger colonies consume and use less energy than their counterparts in smaller colonies, just as the cells of a horse consume and use less energy than the cells of a mouse.
Combining data from actual ant colonies with predictions based on mathematical models, the researchers found that body mass and metabolic rates increased at a consistent, nearly three-quarter-power scaling rate for worker ants and queen ants alike, as well as for their colonies. Based on these findings, the researchers then developed a mathematical model to predict colony lifespan by linking it with colony size, or mass. They found that the larger colonies tended to live longer and use less energy than smaller colonies.
The research is a continuation of a 2010 study Hou and his colleagues published in the Proceedings of the National Academy of Sciences. In that paper, the researchers introduced the idea that insect societies operate like a single superorganism in terms of their physiology and life history.
Hou is first co-author of the Biology Letters article with Dr. Jonathan Z. Shik of North Carolina State University. Other contributors to the study are Dr. Adam Kay of the University of St. Thomas in St. Paul, Minn.; Dr. Michael E. Kaspari, Presidential Professor of Biology at the University of Oklahoma; and Dr. James F. Gillooly, associate professor of biology at the University of Florida.
Other researchers are taking quarter-power scaling and applying it to entire cities, to determine whether they too function as "superorganisms" in some respects, such as energy usage. For instance, says Hou, Dr. Geoffrey West, a theoretical physicist and past president of the Santa Fe Institute, and his colleagues found that "proxies of city energy usage, such as number of gas stations, total length of electric cables and so on, scale sub-linearly with the size of the city," says Hou.
"Just like the examples of ants and cells, between two similar people, the one living in the bigger city is more energy-efficient," Hou says. "As you can imagine, the number of gas stations per capita is smaller in New York than St. Louis, and smaller in St. Louis than Rolla, which means more people share one gas station in New York than in St. Louis, than in Rolla."
Hou, an expert in animal energetics, applies similar energy scaling laws to study how animals uptake energy during growth and how they allocate that energy to growth, health maintenance and reproduction. Recently, his focus has been on the effect of food restriction on extending the animals' life spans.
Provided by Missouri University of Science and Technology

http://bit.ly/UX323E

Blame bacteria if you start putting on weight

Evidence is mounting for the central role that bacteria play in causing obesity.

WEIGHT gain bugging you? Evidence is mounting for the central role that bacteria play in causing obesity. Liping Zhao and his team at Shanghai Jiao Tong University in China put a morbidly obese man on a diet of whole grains, traditional Chinese medicines, probiotics and non-digestible carbohydrates for 23 weeks. The diet was designed to inhibit the bacteria thought to be associated with weight gain by increasing the pH in the colon. The 175-kilogram volunteer lost 51 kg, despite not exercising. People who have had weight-loss surgery lose on average 49 kg.
To see if the bacteria present also changed, the team looked at what species were prevalent in the volunteer's gut before and after the diet. Before the regime, Enterobacter - a toxin-producing pathogen - was most abundant, accounting for 35 per cent of the gut bacteria. After the diet, it was reduced to undetectable levels.
The researchers fed mice samples of this bacterium from the volunteer's gut to determine whether the pathogen was a cause or a result of his obesity. They found that the mice with the new bacteria gained significantly more weight on a high fat diet than control mice, also on a high fat diet (International Society for Microbial Ecology, doi.org/jz9).
Previous work has shown a link between gut bacteria and obesity, but Zhao describes this as "the last missing piece of evidence that bacteria cause obesity". Treatment with an appropriate diet could be cheaper and more effective than surgery, he says.

http://phys.org/news/2012-12-groundbreaking-smallest-virus-particle-implications.html

Groundbreaking research leads to detection of smallest virus particle, implications for early treatment of disease

City tech professor helps discover groundbreaking virus detection method

Vasily Kolchenko, associate professor of biological sciences at New York City College of Technology (City Tech), is a key player on a research team that recently made a breakthrough with enormous potential significance for the treatment of serious diseases.

Their work has made it possible, for the first time, to detect the smallest virus particle. Since even one viral particle can represent a deadly threat, the research likely will make an important contribution to ongoing research on early detection of such diseases as AIDS and cancer.

Until the research team announced their discovery this year in Applied Physics Letters (July 27, 2012), no instrument or methodology had been successful in reliably and accurately detecting a single virus particle, which is in the size range of a nanoparticle. (About 80,000 nanoparticles side by side would have the same width as a human hair.)

The research will potentially have an immense impact on the general public, aiding disease detection at its earliest stage when fewer pathogens are present and medical intervention can be most effective. This new approach also has possible applications in the identification of numerous molecules, especially proteins, which are important for drug development research, both as the targets and the treatments.

While scientists have long used microscopes to view objects as small as bacteria, viruses are much smaller. Even the most sensitive electron microscopes, which are cumbersome, expensive and difficult to operate, cannot guarantee detection of these tiny particles.

The team's breakthrough involved adding a nano-antenna to the light-sensing device to enhance the signal. "The idea that light can 'sense' the presence of nanoparticles and respond to their arrival was groundbreaking," Dr. Kolchenko says. "Since all the deadliest viruses and most interesting biological molecules – proteins and DNA - belong to the nano world, our research proved truly innovative, and its promise is almost unlimited in terms of detecting pretty much everything of interest in life sciences," he adds.

Dr. Kolchenko, who has a medical degree, a doctorate in physiology and a master's degree in mathematics from Kiev University, provided a unique combination of expertise in bioinformatics, mathematics and medicine that was integral to the project's success in isolating the smallest individual RNA virus, MS2.

"I first became interested in pursuing research on using light for the detection and measurement of the tiniest biological and non-living objects when I heard a talk on biosensors that Professor Stephen Arnold of Polytechnic/NYU gave at City Tech," says Dr. Kolchenko, who teaches biology at City Tech and bioinformatics at Polytechnic.

The two-year research project, funded for $400,000 by the National Science Foundation, has been conducted at Polytechnic/NYU's Micro-Particle Laboratory for BioPhotonics, under the direction of Dr. Stephen Arnold, in collaboration with the physics departments of Fordham University and Hunter College, and the biological sciences department of City Tech. Polytechnic/NYU has applied for a utility patent for the team's ground-breaking innovation.

Prior to the latest NSF project, ten years of laboratory research by Dr. Kolchenko and his colleagues resulted in the development of a simple, low-cost design for more sensitive, miniature devices that could detect and measure viruses, proteins and DNA in real time. From 2005 through 2008, the team published papers detailing its progress in such prestigious journals as Applied Physics Letters, Faraday Discussions and Proceedings of the National Academy of Sciences.

"One of the ultimate goals is to develop portable, inexpensive, easy to use and highly sensitive devices for healthcare and research settings," says Dr. Kolchenko. "This research opens the door for highly sensitive detection and measurement of biological and other nanoparticles that are essential in molecular biology, clinical medicine and diagnostics, epidemiology, ecology, nanotechnology and other fields."

Further research is planned, according to Dr. Kolchenko. "Since single protein molecules are much smaller than viral particles, their detection will be the ultimate test of the method," he says. "We hope after some additional research and development, our method will allow for single protein detection as well."

Such research could enable the earlier screening of cancer markers, which are protein molecules produced when cancer grows. Currently, there are several markers that could be potentially detected by the new biosensor; early detection of these markers could allow treatment to begin sooner, enhancing cancer survival rates.

Says Dr. Kolchenko, "We have merely scratched the surface of what is likely to be possible."

Provided by The City University of New York

http://phys.org/news/2012-12-satire-american-citizens.html

Satire is shaping the next generation of American citizens

Satire has always played an important role in democracy, but acurrent group of television satirists are more influential than ever with American citizens, particularly younger ones, according to a Penn State researcher.

Phys.org - Sophia McClennen, professor of international affairs and comparative literature, said that Stephen Colbert and Jon Stewart, like Jonathan Swift and Benjamin Franklin before them, use satire and parody to poke fun at politics and society with the hope that the humorincreases awareness and motivates change.

"What satire does is reveal the folly of the human condition and most, but not all, of satire has a political angle to it," said McClennen. "Satire is different from typical political humor because it demands critical reflection on the part of the audience, so the laughter isn't the end of the joke."

McClennen writes in her book, Colbert's America (Palgrave Macmillan, 2012), that Colbert's type of satire is playing an important role in American democracy because, in a media environment with a 24-hour news cycle that increasingly blends news and entertainment to attract viewers, young people are seeking more engaging sources of news and information.

Comedy Central's Colbert Report and Jon Stewart Show are not just popular entertainment shows with the younger generation, according to McClennen, they are also cited by younger viewers as important news sources. She said that the ability of these comedians to entertain, while prompting critical reflection is the key to raising awareness about political and social issues discussed on the shows.

"I think what's happening is, younger views are tuning into the Colbert Report, and then after the show they are going out and actively looking into the issues," said McClennen.

As an example of Colbert's ability to stir activism, she writes that he has instigated campaigns from the silly - naming a Hungarian bridge after himself - to the serious - donating to Japanese tsunami relief - that were embraced by his fans.

McClennen writes that Colbert's show averages more than a million viewers for each episode, and he has more than 1.9 million Twitter followers. In 2008, when Colbert briefly ran for president, he had a million Facebook users sign up to back his campaign.

Colbert does a twist on satire by adding parody to his routine, McClennen said. The comedian portrays a conservative television commentator modeled on Fox News commentator Bill O'Reilly.

"He has to be the thing he doesn't like, the guy he detests," said McClennen. "Colbert is essentially saying, 'you have been suckered into pundit culture, then I'm going to be the biggest pundit there is.'"

Colbert has been able to not only raise awareness, but change the dialogue at times, too. He coined terms like, "truthiness" to describe when people believe something without facts to support it and "wikiality" to describe the ability to edit out parts of reality that people do not like.

McClennen writes that several factors are increasing the importance of the Colbert Report and other showsthat use political satire. The rise of fundamentalist religious groups, theincreasing power of corporations in the media and a deep divide between political parties are harming the democratic process in the country, McClennen said. "These are harming the democracy in a pretty direct way," she said.

Provided by Pennsylvania State University

http://bit.ly/12BOv37

Human hands evolved so we could punch each other

Forget toolmaking, think fisticuffs. Did evolution shape our hands not for dexterity but to form fists so we could punch other people?

23:00 19 December 2012 by Sara Reardon

That idea emerges from a new study, although it runs counter to conventional wisdom.

About the same time as we stopped hanging from trees and started walking upright, our hands become short and square, with opposable thumbs. These anatomical changes are thought to have evolved for tool manipulation, but David Carrier at the University of Utah in Salt Lake City has an alternative explanation.

He says there are several possible hand shapes that would have allowed greater dexterity, making it less clear why we ended up with the hands we have. But only one hand shape lets us make a fist with a thumb as buttress.

Among primates' hands, ours is unique for its ability to form a fist with the thumb outside the fingers. The fingers of other primates' hands are too long to curl into their palms, and their thumbs are too short to reach across the fingers. So when apes fight, they are far more likely to wrestle or hold their opponent down while others stomp on him, says Carrier.

To test the importance of fists, Carrier and his colleagues recruited 10 athletes and measured how hard they could hit a punching bag using a normal fist, a fist with the thumb stuck out, and with an open palm.

The athletes could generate more than twice the force with a normal fist as with the thumb-stuck-out fist, because of thumb's buttressing role. There was no difference in the force they could generate with a normal fist and with an open palm, but Carrier says it's possible that a fist concentrates the force into a smaller area and so does more damage.

Cause or effect?

Mary Marzke of Arizona State University in Tempe says the study is interesting, but it far from proves that the ability to make a strong fist was the main driver behind the evolution of our hands' shape. It is more likely that it was a useful side effect of a whole suite of modifications.

She points out that apes strike with the heel of their hand when knocking fruit out of trees. Carrier's study didn't assess the force that the heel of the hand generates, but if it turns out to be as good as a fist, it becomes less clear that our hands evolved so as to be perfect for fist-making, Marzke says.

But if the hypothesis is true, Carrier thinks it could explain another mystery. It has long been unclear why high levels of testosterone cause men's ring fingers to be longer than their index fingers. He says the finger-length ratio makes sense if it generates a better fist. This would make dominant males even better fighters.

Journal reference: Journal of Experimental Biology, doi:10.1242/jeb.075713

http://www.eurekalert.org/pub_releases/2012-12/ucl-ool121912.php

Origin of life emerged from cell membrane bioenergetics

A coherent pathway which starts from no more than rocks, water and carbon dioxide and leads to the emergence of the strange bio-energetic properties of living cells, has been traced for the first time in a major hypothesis paper in Cell this week.

At the origin of life the first protocells must have needed a vast amount of energy to drive their metabolism and replication, as enzymes that catalyse very specific reactions were yet to evolve. Most energy flux must have simply dissipated without use. So where did it all that energy come from on the early Earth, and how did it get focused into driving the organic chemistry required for life?

The answer lies in the chemistry of deep-sea hydrothermal vents. In their paper Nick Lane (UCL, Genetics, Evolution and Environment) and Bill Martin (University of Dusseldorf) address the question of where all this energy came from - and why all life as we know it conserves energy in the peculiar form of ion gradients across membranes. "Life is, in effect, a side-reaction of an energy-harnessing reaction. Living organisms require vast amounts of energy to go on living," said Nick Lane.

Humans consume more than a kilogram (more than 700 litres) of oxygen every day, exhaling it as carbon dioxide. The simplest cells, growing from the reaction of hydrogen with carbon dioxide, produce about 40 times as much waste product from their respiration as organic carbon (by mass). In all these cases, the energy derived from respiration is stored in the form of ion gradients over membranes.

This strange trait is as universal to life as the genetic code itself. Lane and Martin show that bacteria capable of growing on no more than hydrogen and carbon dioxide are remarkably similar in the details of their carbon and energy metabolism to the far-from-equilibrium chemistry occurring in a particular type of deep-sea hydrothermal vent, known as alkaline hydrothermal vents. Based on measured values, they calculate that natural proton gradients, acting across thin semi-conducting iron-sulfur mineral walls, could have driven the assimilation of organic carbon, giving rise to protocells within the microporous labyrinth of these vents.

They go on to demonstrate that such protocells are limited by their own permeability, which ultimately forced them to transduce natural proton gradients into biochemical sodium gradients, at no net energetic cost, using a simple Na+/H+ transporter. Their hypothesis predicts a core set of proteins required for early energy conservation, and explains the puzzling promiscuity of respiratory proteins for both protons and sodium ions.

These considerations could also explain the deep divergence between bacteria and archaea (single celled microorganisms) . For the first time, says Lane, "It is possible to trace a coherent pathway leading from no more than rocks, water and carbon dioxide to the strange bioenergetic properties of all cells living today."

The study was funded by a UCL Provost's Venture Research Fellowship, the Leverhulme Trust and the European Research Council.

http://www.eurekalert.org/pub_releases/2012-12/uoc-wcm121312.php

Wallace's century-old map of natural world updated

Until today, Alfred Russell Wallace's century old map from 1876 has been the backbone for our understanding of global biodiversity.

Thanks to advances in modern technology and data on more than 20,000 species, scientists from University of Copenhagen have now produced a next generation map depicting the organisation of life on Earth. Published online in Science Express today, the new map provides fundamental information regarding the diversity of life on our planet and is of major significance for future biodiversity research.

An essential question in understanding life on Earth is why species are distributed the way they are across the planet. This new global map shows the division of nature into 11 large biogeographic realms and shows how these areas relate to each other. It is the first study to combine evolutionary and geographical information for all known mammals, birds and amphibians, a total of over 20,000 species.

Based on the work at the Center for Macroecology, Evolution and Climate at the University of Copenhagen involving 15 international researchers and 20 years of data compilation, the study is published today in Science Express.

Map of terrestrial zoogeographic regions of the world published in "The Geographic Distributions of Animals" (1876) by Alfred Russell Wallace.

The first attempt to describe the natural world in an evolutionary context was made in 1876 by Alfred Russel Wallace, the co-discoverer of the theory of natural selection, along with Charles Darwin. "Our study is a long overdue update of one of the most fundamental maps in natural sciences. For the first time since Wallace's attempt we are finally able to provide a broad description of the natural world based on incredibly detailed information for thousands of vertebrate species," says co-lead-author, Dr. Ben Holt from the Center for Macroecology, Evolution and Climate.

The new map can be split into finer geographical details for each class of animals. It is made freely available to contribute to a wide range of biological sciences, as well as conservation planning and management of biodiversity.

***Updated map by CMEC* Journal Science / AAAS**

Hundreds of thousands of records

Modern technology like DNA sequencing and a tremendous compilation of hundreds of thousands of distribution records on mammals, birds and amphibians across the globe has made it possible to produce the map.

"The map provides important baseline information for future ecological and evolutionary research. It also has major conservation significance in light of the on-going biodiversity crisis and global environmental change. Whereas conservation planners have been identifying priority areas based on the uniqueness of species found in a given place, we can now begin to define conservation priorities based on millions of years of evolutionary history," says Dr. Jean-Philippe Lessard, the other co-lead-author from the Copenhagen center, who is currently based at McGill University, Canada.

Senior author Carsten Rahbek, director of the Center for Macroecology, Evolution and Climate adds: "Despite the incredible advances of natural science, we are still battling to understand the underlying laws that govern life on the planet. This holistic description of the natural world that we provide could be a new cornerstone in fundamental biology."

http://www.eurekalert.org/pub_releases/2012-12/foas-sdk122012.php

Stroke drug kills bacteria that cause ulcers and tuberculosis

New research in the FASEB Journal shows that a compound called ebselen kills bacteria through the inhibition of thioredoxin reductase

Bethesda, MD - A drug currently being used to treat ischemic strokes may prove to be a significant advance in the treatment of tuberculosis and ulcers. In a new research report appearing online in The FASEB Journal, a compound called ebselen effectively inhibits the thioredoxin reductase system in a wide variety of bacteria, including Helicobacter pylori which causes gastric ulcers and Mycobacterium tuberculosis which causes tuberculosis. Thioredoxin and thioredoxin reductase proteins are essential for bacteria to make new DNA, and protect them against oxidative stress caused by the immune system. Targeting this system with ebselen, and others compounds like it, represents a new approach toward eradicating these bacteria.

"This new antibacterial principle provides better chances of surviving an infection," said Arne Holmgren, M.D., Ph.D., a researcher involved in the work from the Division of Biochemistry in the Department of Medical Biochemistry and Biophysics, at Karolinska Institutet in Stockholm, Sweden. "Since ebselen is also an

antioxidant, the present mechanism can be described as a 'two for the price of one' antioxidant action in inflammation, and specific targeting of multi-resistant bacterial complications and sepsis."
Building on previous observations where ebselen has shown antibacterial properties against some bacteria, Holmgren and colleagues hypothesized that the bacteria sensitive to ebselen relied solely on thioredoxin and thioredoxin reductase for essential cellular processes. They investigated this by testing it on strains of E. coli with deletions in the genes for thioredoxin, thioredoxin reductase and the glutaredoxin system. They found that strains with deletions in the genes coding for glutaredoxin system were much more sensitive than normal bacteria. Researchers further tested ebselen against Helicobacter pylori and Mycobacterium tuberculosis, which both naturally lack the glutaredoxin system and are frequently resistant to many commonly used antibiotics, and found both to be sensitive to ebselen.
"As rapidly as these organisms evolve, we need new drugs sooner rather than later," said Gerald Weissmann, M.D., Editor-in-Chief of The FASEB Journal. "The fact that these scientists have found a new target for killing some of the most resistant bacteria is great news, but the fact that we already have at least one drug which we could possibly use now makes the news even better."
Jun Lu, Alexios Vlamis-Gardikas, Karuppasamy Kandasamy, Rong Zhao, Tomas N. Gustafsson, Lars Engstrand, Sven Hoffner, Lars Engman, and Arne Holmgren. Inhibition of bacterial thioredoxin reductase: an antibiotic mechanism targeting bacteria lacking glutathione. FASEB J doi:10.1096/fj.12-223305 ; http://www.fasebj.org/content/early/2012/12/17/fj.12-223305.abstract

http://phys.org/news/2012-12-team-rare-meteorite-possibly-outer.html

Team studies rare meteorite possibly from the outer asteroid belt

Scientists found treasure when they studied a meteorite recovered at Sutter's Mill

Phys.org - Scientists found treasure when they studied a meteorite that was recovered April 22, 2012 at Sutter's Mill, the gold discovery site that led to the 1849 California Gold Rush. Detection of the falling meteorites by Doppler weather radar allowed for rapid recovery so that scientists could study for the first time a primitive meteorite with little exposure to the elements, providing the most pristine look yet at the surface of primitive asteroids. An international team of 70 researchers reported in today's issue of Science that this meteorite was classified as a Carbonaceous-Mighei or CM-type carbonaceous chondrite and that they were able to identify for the first time the source region of these meteorites.
"The small three meter-sized asteroid that impacted over California's Sierra Nevada came in at twice the speed of typical meteorite falls," said lead author and meteor astronomer Peter Jenniskens of the SETI Institute, Mountain View, Calif., and NASA Ames Research Center, Moffett Field, Calif. "Clocked at 64,000 miles per hour, it was the biggest impact over land since the impact of the four meter-sized asteroid 2008 TC3, four years ago over Sudan."
The asteroid approached on an orbit that still points to the source region of CM chondrites. From photographs and video of the fireball, Jenniskens calculated that the asteroid approached on an unusual low-inclined almost comet-like orbit that reached the orbit of Mercury, passing closer to the sun than known from other recorded meteorite falls.
"It circled the sun three times during a single orbit of Jupiter, in resonance with that planet," Jenniskens said. Based on the unusually short time that the asteroid was exposed to cosmic rays, there was not much time to go slower or faster around the sun. That puts the original source asteroid very close to this resonance, in a low inclined orbit. "A good candidate source region for CM chondrites now is the Eulalia asteroid family, recently proposed as a source of primitive C-class asteroids in orbits that pass Earth," adds Jenniskens.
New meteorite suggests that asteroid surfaces more complex than previously thought
After the asteroid broke up in the atmosphere, weather radar briefly detected a hailstorm of falling meteorites over the townships of Coloma and Lotus in California. This enabled a rapid recovery that permitted the most pristine look yet at a CM-type carbonaceous chondrite.
"This was the first time a rare carbonaceous chondrite meteorite was recovered based on such weather radar detection," said Marc Fries of the Planetary Science Institute in Tucson, Arizona, who pioneered the use of this technique. "Meteorites were found mostly under the radar footprint."
Of the estimated 100,000-pound asteroid, less than two pounds was recovered on the ground in the form of 77 meteorites. The biggest was 205 grams. Some of the key meteorites discussed in this work were found by volunteer search teams led by Jenniskens. "The entire Ames community really came together in the search for these meteorites. People work at NASA because they love science and that was very evident when we saw the overwhelming response of volunteers from Ames wanting to be a part of this," said Pete Worden, director of NASA Ames Research Center.

"The meteorite was a jumbled mess of rocks, called a regolith breccia, that originated from near the surface of a primitive asteroid," said meteoriticist Derek Sears of NASA Ames.
NASA and the Japanese space agency (JAXA) have plans to target asteroids similar to the one recovered at Sutter's Mill. The Sutter's Mill meteorite provides a rare glimpse of what these space missions may find.
"NASA's robotic OSIRIS-REx mission is currently being prepared to bring back a pristine sample of an asteroid named 1999 RQ36," said co-author and mission co-investigator Scott Sandford of NASA Ames. "In addition, Sutter's Mill has the same reflective properties as near-Earth asteroid, 1999 JU3, the mission target of the Hayabusa 2 sample return mission currently being prepared by the Japanese space agency, JAXA."
The rapid recovery resulted in the detection of compounds that quickly disappear once a meteorite lands on Earth. Mike Zolensky, a mineralogist at NASA's Johnson Space Flight Center, Houston, was surprised to detect the mineral oldhamite, a calcium sulfide, known in the past to disappear from contact with water by simply breathing on it. "This mineral was known before mainly from rare enstatite chondrites," said Zolensky, "and its presence in the regolith breccia could mean that primitive and highly evolved asteroids collided with each other even at early times when the debris accumulated that now makes the meteorite matrix."
A wide array of carbon-containing compounds was detected that quickly reacted with water once in the Earth's environment. It is thought that the carbon atoms in our body may have been brought to Earth by such primitive asteroids in the early stages of our planet's history.
"Amino acids were few in this meteorite because this particular meteorite appears to have been slightly heated in space before it arrived at Earth," said Danny Glavin of NASA's Goddard Space Flight Center, Greenbelt, Md. It appears that different parts of the meteorite had a different thermal alteration history. Heating also removed some of the water that used to move salts around in the asteroid.
"Samples collected before it rained on the meteorite fall area still contained such salts," said George Cooper of NASA Ames, "but Sutter's Mill was less altered by water in the asteroid itself than other CM type meteorites."
"Only 150 parts per billion of Sutter's Mill was actual gold," said co-author and cosmochemist Qing-zhu Yin of U.C. Davis, Davis, Calif., "but all of it was scientific gold. With 78 other elements measured, Sutter's Mill provides one of the most complete records of elemental compositions documented for such primitive meteorites."
More information: "Radar-Enabled Recovery of the Sutter's Mill Meteorite, a Carbonaceous Chondrite Regolith Breccia," by P. Jenniskens, Science paper: DOI 10.1126/science.1227163 dx.doi.org/10.5531/sd.eps.1 Provided by Field Museum

http://www.sciencedaily.com/releases/2012/12/121220144124.htm

Major Source of Evolutionary Differences Among Species Uncovered

University of Toronto Faculty of Medicine researchers have uncovered a genetic basis for fundamental differences between humans and other vertebrates that could also help explain why humans are susceptible to diseases not found in other species.

Scientists have wondered why vertebrate species, which look and behave very differently from one another, nevertheless share very similar repertoires of genes. For example, despite obvious physical differences, humans and chimpanzees share a nearly identical set of genes.
The team sequenced and compared the composition of hundreds of thousands of genetic messages in equivalent organs, such as brain, heart and liver, from 10 different vertebrate species, ranging from human to frog. They found that alternative splicing -- a process by which a single gene can give rise to multiple proteins -- has dramatically changed the structure and complexity of genetic messages during vertebrate evolution.
The results suggest that differences in the ways genetic messages are spliced have played a major role in the evolution of fundamental characteristics of species. However, the same process that makes species look different from one another could also account for differences in their disease susceptibility.
"The same genetic mechanisms responsible for a species' identity could help scientists understand why humans are prone to certain diseases such as Alzheimer's and particular types of cancer that are not found in other species," says Nuno Barbosa-Morais, the study's lead author and a computational biologist in U of T Faculty of Medicine's Donnelly Centre for Cellular and Biomolecular Research. "Our research may lead to the design of improved approaches to study and treat human diseases."
One of the team's major findings is that the alternative splicing process is more complex in humans and other primates compared to species such as mouse, chicken and frog.
"Our observations provide new insight into the genetic basis of complexity of organs such as the human brain," says Benjamin Blencowe, Professor in U of T's Banting and Best Department of Research and the Department of Molecular Genetics, and the study's senior author.
"The fact that alternative splicing is very different even between closely related vertebrate species could ultimately help explain how we are unique."

http://www.scientificamerican.com/podcast/episode.cfm?id=norovirus-survives-restaurant-dishw-12-12-20

Restaurant Dishwashing Protocols

Dishwashing protocols designed with food-borne bacteria in mind didn't cut the mustard in a test with noroviruses, which cause nasty gastrointestinal illnesses. Karen Hopkin reports

Download MP3

Thinking of eating out over the holidays? Well, you might consider bringing your own plate. Because a new study shows that the typical dishwashing procedures used by most restaurants can't wash away viruses that can cause some serious stomach distress. Those unsavory findings are served up in the journal PLoS ONE. [Lizanel Feliciano et al., Efficacies of Sodium Hypochlorite and Quaternary Ammonium Sanitizers for Reduction of Norovirus and Selected Bacteria during Ware-Washing Operations]

Food poisoning is no fun. And one potential source of sickness when you dine out is the dinnerware. Plates, forks, and glasses could transfer a bug from contaminated foodstuff or a previous diner to you. Most public eateries follow certain industry guidelines when it comes to cleaning their tableware. But those protocols were designed with foodborne bacteria in mind. So what happens if the infectious agent is viral?

To find out, researchers inoculated cream cheese and milk with either E. coli bacteria or a norovirus, famous for spreading throughout cruise ships. The researchers shmeared the mess over ceramic plates, utensils and glassware…and then put the dirty dishes in the wash. And they found that while the standard detergents did a decent job at sluicing away the E. coli, they didn't get rid of the dairy-encrusted virus.

So bon appetit. And caveat emptor.

http://www.bbc.co.uk/news/health-20795977

Trojan-horse therapy 'completely eliminates' cancer in mice

An experimental "Trojan-horse" cancer therapy has completely eliminated prostate cancer in experiments on mice, according to UK researchers.

By James Gallagher Health and science reporter, BBC News

The team hid cancer killing viruses inside the immune system in order to sneak them into a tumour.

Once inside, a study in the journal Cancer Research showed, tens of thousands of viruses were released to kill the cancerous cells. Experts labelled the study "exciting," but human tests are still needed.

Using viruses to destroy rapidly growing tumours is an emerging field in cancer therapy, however one of the challenges is getting the viruses deep inside the tumour where they can do the damage. "There's a problem with getting enough virus into the tumour," Prof Claire Lewis from the University of Sheffield told the BBC. She leads a team which uses white blood cells as 'Trojan horses' to deliver the viral punch.

'Surfing'

After chemotherapy or radiotherapy is used to treat cancer, there is damage to the tissue. This causes a surge in white blood cells, which swamp the area to help repair the damage. "We're surfing that wave to get as many white blood cells to deliver tumour-busting viruses into the heart of a tumour," said Prof Lewis.

Her team takes blood samples and extract macrophages, a part of the immune system which normally attacks foreign invaders. These are mixed with a virus which, just like HIV, avoids being attacked and instead becomes a passenger in the white blood cell.

In the study, the mice were injected with the white blood cells two days after a course of chemotherapy ended. At this stage each white blood cell contained just a couple of viruses. However, once the macrophages enter the tumour the virus can replicate. After about 12 hours the white blood cells burst and eject up to 10,000 viruses each - which go on to infect, and kill, the cancerous cells.

Gone

At the end of the 40-day study, all the mice who were given the Trojan treatment were still alive and had no signs of tumours. By comparison, mice given other treatments died and their cancer had spread. Prof Lewis said: "It completely eradicates the tumour and stops it growing back." She said it was a "ground-breaking" concept, but cautioned that many remarkable advances in treating mice failed to have any effect in people. She hopes to begin human trials next year.

Dr Emma Smith, from Cancer Research UK, said: "Harnessing the body's own immune system to deliver a deadly virus to tumours is an exciting approach that many scientists are pursuing. "This study shows it has the potential to make chemotherapy and radiotherapy more effective weapons against cancer. She said the research was still at an early stage and tests to show it is safe and effective in humans are still needed.

Dr Kate Holmes, head of research at Prostate Cancer UK, said: "It demonstrates that this innovative method of delivering a tumour-killing virus direct to the cancer site is successful at reducing the development of advanced prostate tumours in mice which have been treated with chemotherapy and radiotherapy.

"If this treatment goes on to be successful in human trials, it could mark substantial progress in finding better treatments for men with prostate cancer which has spread to the bone."

http://www.nasa.gov/topics/solarsystem/features/watchtheskies/quadrantids_2013.html

Quadrantids create year's first meteor shower

A little-known meteor shower named after an extinct constellation, the Quadrantids will present an excellent chance for hardy souls to start the year off with some late-night meteor watching.

Peaking in the wee morning hours of Jan. 3, the Quadrantids have a maximum rate of about 80 per hour, varying between 60-200. Unfortunately, light from a waning gibbous moon will wash out many Quadrantids, cutting down on the number of meteors seen by skywatchers.

Unlike the more famous Perseid and Geminid meteor showers, the Quadrantids only last a few hours, so it's the morning of Jan. 3 or nothing. Given the location of the radiant - northern tip of Bootes the Herdsman - only observers at latitudes north of 51 degrees south will be able to see Quadrantids.

Like the Geminids, the Quadrantids originate from an asteroid, called 2003 EH1. Dynamical studies suggest that this body could very well be a piece of a comet which broke apart several centuries ago, and that the meteors you will see before dawn on Jan. 3 are the small debris from this fragmentation. After hundreds of years orbiting the sun, they will enter our atmosphere at 90,000 mph, burning up 50 miles above Earth's surface - a fiery end to a long journey!

Watch the Quadrantids! Live Ustream Feed

A live Ustream feed of the Quadrantid shower will be embedded below on the nights of Jan. 2-4. The camera is mounted at NASA's Marshall Space Flight Center in Huntsville, Ala. During the day you will see either pre-recorded footage or a blank box - the camera is light-activated and turns on at dusk.

Do You Have Some Great Quadrantid Images?

If you have some great images of the Quadrantid meteor shower, please consider adding them to the Quadrantid Meteors photo group in Flickr. Who knows - your images may attract interest from the media and receive international exposure.

More About the Quadrantids

The Quadrantids derive their name from the constellation of Quadrans Muralis (mural quadrant), which was created by the French astronomer Jerome Lalande in 1795. Located between the constellations of Bootes and Draco, Quadrans represents an early astronomical instrument used to observe and plot stars. Even though the constellation is no longer recognized by astronomers, it was around long enough to give the meteor shower - first seen in 1825 - its name.

http://slate.me/V6qFpT

Dengue, aka "Breakbone Fever," Is Back

The vicious virus has re-established itself in the South, and mosquitoes are carrying it north.

By Maryn McKenna|Posted Friday, Dec. 21, 2012, at 11:14 AM ET

In the autumn of 1885, people in Austin, Texas, began to feel sick. One after another, they developed a chill and then a soaring fever. They vomited and broke out in rashes. Their most distinctive symptom was agonizing pain behind their eyes and in the bones of their arms and legs. And when the fever subsided, lack of appetite and deep exhaustion left them unable to work for weeks or months.

Austin had been founded only 46 years before, and it was still small, with just 22,000 people. By the time the epidemic was over, 16,000 of them had fallen ill. A local doctor who described the outbreak in the Journal of the American Medical Association the following year added: "I am informed that other cities ... had as many cases in proportion to the population as did Austin."

The illness that took out Texas that fall had already devastated Charleston, S.C., in 1828 and Savannah, Ga., in 1850, and it would go on to sicken half the population of Galveston, Texas, in 1897; one-quarter of Monroe, La., in 1922; and one out of every nine people in Miami in 1934. It was dengue - a mosquito-borne virus popularly known as "breakbone fever" for the pain it caused. From the 1820s to the 1940s, it caused recurring epidemics roughly every 10 years.

World War II mosquito-eradication programs broke the chain of transmission between humans and insects, and by the time the war ended, dengue had retreated to the tropics and was no longer a problem in the United States. That may be about to change. At the annual meeting of the American Society of Tropical Medicine and Hygiene last month, researchers from the University of Florida revealed that dengue has reappeared in Key West, Fla. The virus they found was not a one-time visitor imported by a tourist or a stray mosquito; it has been on the island long enough to become a genetically distinct, local strain.

The Florida researchers didn't want to talk about their presentation because they hope to get it published soon in a medical journal. But it turns out other tropical-disease experts have been watching dengue's return to the United States for a while and wondering what it will mean.
"It really is just a matter of time until dengue re-establishes itself in certain areas here," says Amesh Adalja of the Center for Biosecurity of the University of Pittsburgh Medical Center. "The U.S. has been lucky that it has escaped so far."
Dengue is already a pandemic elsewhere. Among insect-borne diseases, malaria gets the headlines: It causes about 219 million cases per year and about 660,000 deaths. But dengue is right behind it, racking up potentially 100 million infections per year around the world and putting about 500,000 people in the hospital, most of them children. It causes fewer deaths - 25,000 per year - than malaria, but its prolonged illness keeps people from working and depresses both personal incomes and gross national product.
Dengue is also becoming more common. Between World War II and about 1970, severe dengue epidemics were recorded in only nine tropical countries; now the disease occurs routinely in more than 100. The primary driver has been the growth of slums as people leave rural areas to search for work. When migrants settle at the fringes of a city, they are beyond the reach of its infrastructure - water lines, sewer systems, and trash disposal - and they cope by digging latrines, storing water in jugs and barrels, and consigning trash to open dumps. All of those strategies create small pools of stagnant liquid, exactly the kind of habitat that the main dengue-carrying mosquitoes prefer. (The pools can be very small, less than an ounce.)
Dengue infects only humans and other primates - there is no intermediate host that harbors it, such as birds for West Nile virus - and people are its main vehicle for moving around the globe. After a bite, the virus replicates in the blood for four to seven days; once the fever starts, there are at least two and up to 10 days when the victim can cause an infection in the next mosquito that bites him or her. In the two weeks between the initial bite and the end of the infectious stage, a traveler can unknowingly transport the virus from an area where it is common - a marketplace in Singapore, a river terrace in Thailand, a beach in the Caribbean - to somewhere it has never been before.
The Centers for Disease Control and Prevention estimates that more than 2,700 people between 1977 and 1995, and more than 360 between 2001 and 2004, had that experience: being bitten somewhere, coming home, and getting sick afterward. In most cases, the disease went no further, but sometimes it kept spreading. There was a 122-person outbreak in Hawaii in 2001, the first time the virus had been seen in the islands since 1944. There were 25 cases in Brownsville, Texas, in 2005 and 90 cases in Key West between 2009 and 2010. In the last case, the outbreak extended over the winter, when cooler temperatures should have knocked out local mosquitoes.
"That was the winter when the H1N1 pandemic flu was circulating, and whatever dengue cases we had would have been masked by that and went unrecognized," says Danielle Stanek of the Florida Department of Health. "When the flu settled down and we realized there were still dengue cases, that was a wake-up call for us."
Local spread of dengue is still happening in Florida. On Key West, 5 percent of people show immunologic evidence of having had a dengue infection, and the disease is found farther north as well. This year, four residents caught "locally acquired" dengue, two in Miami and two near Orlando, Fla. Another 112 were diagnosed with dengue they had caught somewhere else and brought there.
The CDC's experts assume there are more cases that haven't been counted, and not just in Florida. "When you're seeing a patient early on, dengue looks like a lot of other acute (fever-causing) illnesses," says Kay Tomashek, chief of epidemiology in the agency's dengue branch. "If you are a physician in New York and you see a patient with fever, headache, and muscle pain, you might not be thinking about that."
Detecting imported cases is important because the more frequently the disease comes across the border, the more risks from it increase. And not just the risk of catching the disease. There are four types of dengue, distributed unevenly across the tropics and subtropical zone. Becoming infected with any one causes the classic breakbone fever. But if you acquire and recover from one type and then contract a different type even years later, you are more likely to develop the disease's worst version, dengue hemorrhagic fever. DHF disrupts the circulation, sends patients into shock, and kills up to 1 in 5.
The U.S. outbreaks to date, as well as the locally adapted Key West strain, are all caused by the first type, known as DEN-1. But 10 of the imported cases in Florida this year were in tourists from Central and South America, where DEN-2, DEN-3, and DEN-4 circulate as well.
Could more dengue outbreaks happen? To spark one, you need three things. First, imported virus: check. Second, a population with no immunity. The United States has that, since dengue was last widespread in the 1940s. And third, mosquitoes that can transmit it. Those are already widespread.
The spraying campaigns that ended U.S. epidemics of malaria and dengue in the 1940s turned out to be only a temporary solution. National eradication programs petered out in 1972, and the main dengue vector, Aedes

aegypti, quickly returned; it is now in 23 states and ranges as far north as New York City. In 1985, a second species that can spread dengue - Aedes albopictus, better known as the Asian tiger mosquito - arrived in Texas in a shipment of used tires from the Pacific Rim that had been stored outdoors and held puddles of rainwater. It is now in 26 states and has been found as far north as Chicago. A. albopictus is what entomologists call a "less competent" vector; it doesn't spread the disease as efficiently as A. aegypti does. But it has other abilities that have huge significance for disease transmission: It bites all day long, not just at dawn and dusk, and it can survive both winter temperatures and drought.

Because there is no vaccine for dengue, the best hope of stopping its advance relies on individual action, such as getting people to wear repellent and persuading them to scour their homes and properties for small puddles - underneath a planter, inside a tiki torch - after every rain. Or convincing them to stay inside. Researchers theorize the 2005 Brownsville outbreak was smaller than the 2009 Key West because of the "Texas lifestyle" of sealed, air-conditioned houses - so different from the patio culture of Hawaii and Key West.

It's impossible to say, at this point, if climate change will move the risks of dengue farther north. Researchers disagree on whether higher temperatures automatically mean bigger mosquito populations, since the insects are also affected by unpredictable changes in rainfall, humidity, and wind. But barring some other factor that no one can foresee, the experts agree: Dengue is coming.

"It may not swamp the entire U.S.," Adalja acknowledged. "But the entire South already harbors those mosquitoes, and that is bad enough. Dengue shouldn't have to swamp the entire country for us to make it a priority."

http://www.sciencedaily.com/releases/2012/12/121221233118.htm

Why and How Patients With Lung Cancer Initially Get Diagnosed With the Disease

Without screening, why and how do patients with lung cancer get diagnosed with the disease

UT Southwestern Medical Center researchers are looking into the widespread implementation of computed-tomography (CT) scanning for the early detection of lung cancer in a public heath setting, asking two key questions: Without screening, why and how do patients with lung cancer get diagnosed with the disease in the first place? And what proportion of these cases would be captured by screening efforts?

Dr. David Gerber, an oncologist and assistant professor of internal medicine, has used the electronic medical records data of more than 400 patients in a single-center study that is further exploring results from the National Lung Screening Trial (NLST) released in 2010. The NLST already showed a reduction in lung cancer mortality may result in widespread CT-based screening of select populations. This population was strictly defined according to age (55 to 74 years) and smoking history (at least 30 years of one-pack-a-day smoking).

Dr. Gerber and his team, in a study published December 22 at PLoS ONE -- the Public Library of Science's online journal, reviewed the records of patients who were diagnosed with Stage 1 or Stage 2 non-small cell lung cancer over a recent 10-year period, and found that the proportion of cases identified by CT scan (without preceding chest X-ray) increased almost 50 percent during this period. Simultaneously, the proportion of patients who underwent initial chest imaging to evaluate symptoms declined more than 30 percent. Finally, the researchers found that only half of early-stage lung cancer cases would meet NLST criteria for lung cancer screening.

"Our results suggest that a substantial proportion of patients currently presenting with early-stage lung cancer would continue to do so independently of radiographic screening if such a program were implemented according to NLST criteria," Dr. Gerber said. "The possibility of frequent detection of early-stage disease outside of a screening context seems more likely with lung cancer than with other malignancies, as chest imaging is a more common practice in non-screening clinical care than are mammograms, Pap smears, and colonoscopies."

Whether radiographic screening for lung cancer should be extended to a broader population is not yet known. "Adhering to specific guidelines may be challenging for clinicians," Dr. Gerber said. "For instance, in our sample, almost 25 percent of patients with early-stage disease would be ineligible for screening because they are too old under NLST criteria."

Dr. Gerber pointed out that certain professional organizations, such as the National Comprehensive Cancer Network, have omitted a maximum age cut-off from their screening recommendations.

"Until there is sufficient evidence to offer screening to a broader population," Dr. Gerber said, "clinicians should remain aware of the diverse reasons for and circumstances of early-stage lung cancer presentation to expedite further evaluation and potentially curative treatment."

Other UT Southwestern researchers involved in the study are lead author Dr. Evelyn O. Taiwo, a former hematology-oncology fellow who is now at the State University of New York Downstate Medical Center; Dr.

Jeffrey T. Yorio, a former resident of internal medicine who is now a fellow at UT M.D. Anderson Cancer Center; and Jingsheng Yan, biostatistical consultant III in the Harold C. Simmons Cancer Center.

Visit the Harold C. Simmons Cancer Center to learn more about oncology at UT Southwestern, including highly individualized treatments for cancer at the region's only National Cancer Institute-designated center.

http://www.sciencenews.org/view/generic/id/347241/description/West_Antarctica_warming_fast

West Antarctica warming fast

Temperature record from high-altitude station shows unexpectedly rapid rise

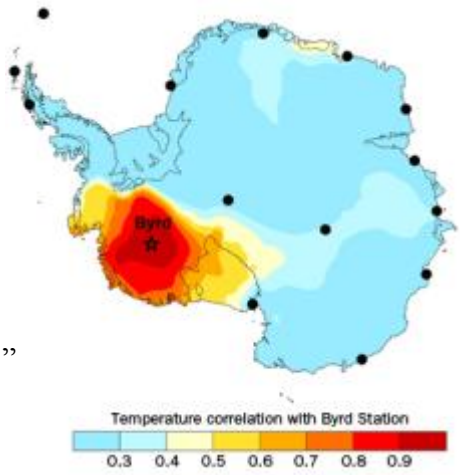
By Alexandra Witze

While the Arctic melts apace with rising global temperatures, Antarctica is often seen as the literal polar opposite - frigid, unyielding, impervious to change. But a spot in the heart of the West Antarctic Ice Sheet is one of the fastest-warming places on Earth, a new study shows.

From 1958 to 2010, the average temperature at the mile-high Byrd station rose by 2.4 degrees Celsius, researchers report online December 23 in Nature Geoscience. That warming is nearly twice what earlier, indirect studies had suggested.

"It's a big number - about as big as the most rapidly warming places elsewhere on the planet," says study coauthor David Bromwich, a polar scientist at Ohio State University in Columbus. "We were quite surprised."

Byrd is warming fastest in winter and spring, but Bromwich and his colleagues also say they detect a statistically significant temperature increase during the summer. If so, then even the frozen Antarctic interior is getting closer to melting.



Red colors indicate parts of Antarctica whose temperatures track closely with those measured at Byrd station (star), where new research shows that temperatures are heating up faster than expected. **Credit: Julien Nicolas/Ohio State**

"The impacts of warming here are potentially huge," says David Schneider, a paleoclimatologist at the National Center for Atmospheric Research in Boulder, Colo. West Antarctica holds far more water locked up as ice than Greenland does, and melting from both great ice sheets has already raised sea levels 11 millimeters over the past two decades (SN: 12/29/12, p. 10).

That's why scientists have been working to tease out whether Antarctica is warming or not. They know that parts of East Antarctica have been cooling, while places along the coast and on the Antarctic peninsula have been warming. But temperature records from the West Antarctic interior are few and far between.

The U.S. Navy established Byrd station in 1957 as part of the International Geophysical Year, and weather observers measured temperatures there until 1975. The station then fell into disuse and automated weather measurements began in 1980. Because of gaps and changes in the way weather data have been collected, many scientists had written off the Byrd record as too spotty to rely on.

But Bromwich's team wanted to take a second look, since many indirect observations - such as measurements from ice cores and holes in the ice - suggest West Antarctica has indeed been getting warmer (SN: 2/14/09, p. 8). Bromwich and his colleagues, including graduate student Julien Nicolas, carefully stitched together the Byrd temperature data. (It helped that the automated weather station was sent back to Wisconsin in 2011 for an upgrade and a recheck of its instruments.) Then the scientists used a sophisticated computer simulation and further data analysis to fill in the missing temperature observations. "There's no doubt it's better than what was done before," says Nicolas.

What happens at Byrd doesn't stay at Byrd: Temperatures at the station track closely with temperatures over a wide swath of West Antarctica, Bromwich says. That suggests the ice sheet may approach melting much closer to the coast, where the ice extends onto the ocean as floating ice shelves that can destabilize and break apart, as the Larsen B shelf did in 2002. Such collapses contribute to sea level rise.

"The Arctic is receiving a lot of attention right now, as it should," Bromwich says. "But what we are trying to emphasize here is that we need to pay attention to the other end of the Earth as well."

Citations

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Chinese medicine yields secrets to scientists at The Scripps Research Institute

Atomic mechanism of 2-headed molecule derived from Chang Shan, a traditional Chinese herb, is shown in unprecedented detail

LA JOLLA, CA– The mysterious inner workings of Chang Shan - a Chinese herbal medicine used for thousands of years to treat fevers associated with malaria - have been uncovered thanks to a high-resolution structure solved at The Scripps Research Institute (TSRI). Described in the journal Nature this week, the structure shows in atomic detail how a two-headed compound derived from the active ingredient in Chang Shan works. Scientists have known that this compound, called halofuginone (a derivative of the febrifugine), can suppress parts of the immune system - but nobody knew exactly how.

The new structure shows that, like a wrench in the works, halofuginone jams the gears of a molecular machine that carries out "aminoacylation," a crucial biological process that allows organisms to synthesize the proteins they need to live. Chang Shan, also known as Dichroa febrifuga Lour, probably helps with malarial fevers because traces of a halofuginone-like chemical in the herb interfere with this same process in malaria parasites, killing them in an infected person's bloodstream.

"Our new results solved a mystery that has puzzled people about the mechanism of action of a medicine that has been used to treat fever from a malaria infection going back probably 2,000 years or more," said Paul Schimmel, PhD, the Ernest and Jean Hahn Professor and Chair of Molecular Biology and Chemistry and member of The Skaggs Institute for Chemical Biology at TSRI. Schimmel led the research with TSRI postdoctoral fellow Huihao Zhou, PhD.

Halofuginone has been in clinical trials for cancer, but the high-resolution picture of the molecule suggests it has a modularity that would make it useful as a template to create new drugs for numerous other diseases.

The Process of Aminoacylation and its Importance to Life

Aminoacylation is a crucial step in the synthesis of proteins, the end products of gene expression. When genes are expressed, their DNA sequence is first read and transcribed into RNA, a similar molecule. The RNA is then translated into proteins, which are chemically very different from DNA and RNA but are composed of chains of amino acid molecules strung together in the order called for in the DNA.

Necessary for this translation process are a set of molecules known as transfer RNAs (tRNAs), which shuttle amino acids to the growing protein chain where they are added like pearls on a string. But before the tRNAs can move the pearls in place, they must first grab hold of them.

Aminoacylation is the biological process whereby the amino acid's pearls are attached to these tRNA shuttles. A class of enzymes known as aminoacyl-tRNA synthetases is responsible for attaching the amino acids to the tRNAs, and Schimmel and his colleagues have been examining the molecular details of this process for years. Their work has given scientists insight into everything from early evolution to possible targets for future drug development. Over time what has emerged as the picture of this process basically involves three molecular players: a tRNA, an amino acid and the aminoacyl-tRNA synthetase enzyme that brings them together. A fourth molecule called ATP is a microscopic form of fuel that gets consumed in the process.

The new work shows that halofuginone gets its potency by interfering with the tRNA synthetase enzyme that attaches the amino acid proline to the appropriate tRNA. It does this by blocking the active site of the enzyme where both the tRNA and the amino acid come together, with each half of the halofuginone blocking one side or the other.

Interestingly, said Schimmel, ATP is also needed for the halofuginone to bind. Nothing like that has ever been seen in biochemistry before. "This is a remarkable example where a substrate of an enzyme (ATP) captures an inhibitor of the same enzyme, so that you have an enzyme-substrate-inhibitor complex," said Schimmel.

The article, "ATP-Directed Capture of Bioactive Herbal-Based Medicine on Human tRNA Synthetase," by Huihao Zhou, Litao Sun, Xiang-Lei Yang and Paul Schimmel was published in the journal Nature on December 23, 2012

This work was supported by the National Institutes of Health through grants #GM15539, #23562 and #88278 and by a fellowship from the National Foundation for Cancer Research.

http://www.eurekalert.org/pub_releases/2012-12/uoc--ndc122112.php

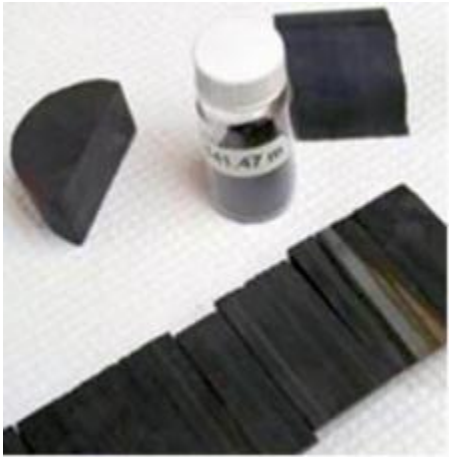
New data challenge old views about evolution of early life

UC Riverside-led research team rules out zinc as a factor in the delayed diversification of single-celled and multicellular organisms

RIVERSIDE, Calif. - A research team led by biogeochemists at the University of California, Riverside has tested a popular hypothesis in paleo-ocean chemistry, and proved it false.

The fossil record indicates that eukaryotes - single-celled and multicellular organisms with more complex cellular structures compared to prokaryotes, such as bacteria - show limited morphological and functional

diversity before 800-600 million years ago. Many researchers attribute the delayed diversification and proliferation of eukaryotes, which culminated in the appearance of complex animals about 600 million years ago, to very low levels of the trace metal zinc in seawater.
As it is for humans, zinc is essential for a wide range of basic cellular processes. Zinc-binding proteins, primarily located in the cell nucleus, are involved in the regulation of gene transcription.
Eukaryotes have increasingly incorporated zinc-binding structures during the last third of their evolutionary history and still employ both early- and late-evolving zinc-binding protein structures. Zinc is, therefore, of particular importance to eukaryotic organisms. And so it is not a stretch to blame the 1-2-billion-year delay in the diversification of eukaryotes on low bioavailability of this trace metal.
But after analyzing marine black shale samples from North America, Africa, Australia, Asia and Europe, ranging in age from 2.7 billion years to 580 million years old, the researchers found that the shales reflect high seawater zinc availability and that zinc concentrations during the Proterozoic (2.5 billion to 542 million years ago) were similar to modern concentrations. Zinc, the researchers posit, was never biolimiting.
Study results appear online Dec. 23 in Nature Geoscience.
"We argue that the concentration of zinc in ancient marine black shales is directly related to the concentrations of zinc in seawater and show that zinc is abundant in these rocks throughout Earth's history," said Clint Scott, the first author of the research paper and a former UC Riverside graduate student. "We found no evidence for zinc biolimitation in seawater."
Scott, now a research geologist with the U.S. Geological Survey, explained that the connection between zinc limitation and the evolution of eukaryotes was based largely on the hypothesis that Proterozoic oceans were broadly sulfidic.
Under broadly sulfidic conditions, zinc should have been scarce because it would have rapidly precipitated in the oceans, he explained.



Organic-rich shale samples, such as these from the 2.5-billion-year-old Mount McRae Shale from Western Australia, were analyzed for their zinc contents. The results confirm that the early ocean was not Zn-lean and that other controls must be invoked to explain the protracted appearance and proliferation of eukaryotic life. **Arizona State University**

"However, a 2011 research paper in Nature also published by our group at UCR demonstrated that Proterozoic oceans were more likely broadly ferruginous - that is, low in oxygen and iron-rich - and that sulfidic conditions were more restricted than previously thought," said Scott, who performed the research in the lab of Timothy Lyons, a professor of biogeochemistry and the principal investigator of the research project.
The research team argues that ferruginous deep oceans, combined with large hydrothermal fluxes of zinc via volcanic activity on the seafloor, maintained high levels of dissolved zinc throughout the oceans and provided a relatively stable marine reservoir of the trace metal over the past 2.7 billion years.
"The key challenge in understanding the early evolution of life is recognizing the environmental conditions under which that life first appeared and diversified," Lyons said.
"We have taken a very direct approach that specifically tracks the availability of essential micronutrients, and, to our surprise, zinc supplies in ancient seawater were much higher and less variable than previously imagined.
"We can imagine for the first time," he quipped, "that zinc supplements were not on the shopping lists of our early eukaryotic ancestors, and so we better find another reason to explain the mysterious delay in their rise in the ocean."

Scott, who graduated with a doctoral degree in geological sciences from UCR in 2009, and Lyons were joined in the study by Noah J. Planavsky, a former UCR graduate student in Lyons' lab; Chris L. Dupont at the J. Craig Venter Institute, La Jolla, Calif.; Brian Kendall and Ariel D. Anbar at Arizona State University; Benjamin C. Gill at Virginia Polytechnic Institute and State University and also a former member of the Lyons lab; Leslie J. Robbins and Kurt O. Konhauser at the University of Alberta, Canada; Kathryn F. Husband and Simon W. Poulton at the University of Leeds, United Kingdom; Gail L. Arnold at the Max Planck Institute for Marine Microbiology, Germany; Boswell A. Wing at McGill University, Canada; and Andrey Bekker at the University of Manitoba, Canada. The idea for the study was a direct consequence of the 2011 Nature paper by Planavsky, Scott, Lyons and others that challenged the hypothesis of broadly sulfidic oceans.

The international collaboration received funding for the study from numerous sources. In the U.S., funding came from the National Science Foundation, the NASA Astrobiology Institute and the Agouron Institute.

http://www.eurekalert.org/pub_releases/2012-12/wt-stp122112.php

Study turns parasite invasion theory on its head

Current thinking on how the Toxoplasma gondii parasite invades its host is incorrect, according to a study published today in Nature Methods describing a new technique to knock out genes.

The findings could have implications for other parasites from the same family, including malaria, and suggest that drugs that are currently being developed to block this invasion pathway may be unsuccessful.

Toxoplasma gondii is a parasite that commonly infects cats but is also carried by other warm-blooded animals, including humans. Up to a third of the UK population are chronically infected with the parasite. In most cases the acute infection causes only flu-like symptoms. However, women who become infected during pregnancy can pass the parasite to their unborn child which can result in serious health problems for the baby such as blindness and brain damage. People who have compromised immunity, such as individuals infected with HIV, are also at risk of serious complication due to reactivation of dormant cysts found in the brain..

Researchers at the Wellcome Trust Centre for Molecular Parasitology at the University of Glasgow made the discovery using a new technique to knock out specific genes in the parasite's genome. They specifically looked at three genes that are considered to be essential for the parasite to invade cells within its host to establish an infection.

"We found that we can remove each of these genes individually and the parasite can still penetrate the host cell, showing for the first time that they are not essential for host cell invasion as was previously thought," said Dr Markus Meissner, a Wellcome Trust Senior Research Fellow who led the study. "This means that the parasite must have other invasion strategies at its disposal that need to be investigated."

The genes the researchers looked at form the core of the parasite's gliding machinery that enable it to move around. In the past, researchers have only ever been able to reduce the expression level of these genes in the parasite, which did lead to a reduction in host cell invasion but invasion was never blocked completely. This was attributed to the low levels of gene expression that persisted. However, with the new technique, the team were able to completely remove the genes of interest. Unexpectedly they found that the parasites were still able to invade.

"One of the genes we looked at is the equivalent of a malaria gene that is a major candidate for vaccine development. Our findings would suggest that such a vaccine may not be successful at preventing malaria infection and we need to revisit our understanding of how this family of parasites invades host cells," added Dr Meissner.

As well as malaria, a number of other parasites that affect livestock also belong to the same family. The findings could also provide clues to new treatments for these diseases, which cause substantial economic losses worldwide.

http://phys.org/news/2012-12-population-growth-consumption-farming-farmlands.html

Changes in population growth, consumption and farming begin to return former farmlands to nature

Analysis of global land use and population growth leads scientists to conclude that use of land for farming has peaked, with former farmlands returning to nature.

Phys.org - With the global population racing past seven billion, demographers and world leaders have been concerned with depletion of resources to support everyone. The future, though, may be less bleak than some have feared. Changes in population growth and how farmers use land have brought the world to "peak farmland," a team of Rockefeller University scientists report in a special issue of the journal Population and Development Review.

"We are excited to report that we believe that humanity has reached peak farmland, and that a large net global restoration of land to nature is ready to begin," says senior author Jesse H. Ausubel, director of Rockefeller's Program for the Human Environment. "Happily, the cause is not exhaustion of arable land, as many have feared, but rather moderation of population and tastes and ingenuity of farmers."

Ausubel, with co-authors Paul Waggoner and Iddo K. Wernick, analyzed factors such as global land use and population growth over the last 50 years. Looking at the production index of all crops of the UN's Food and Agriculture Organization, they found that from 1961 to 2009 land farmed grew by only 12 percent while the index rose about 300 percent.

"Without lifting crop production per hectare, farmers would have needed about 3 billion more hectares, about the sum of the United States, Canada and China, or almost twice South America," says Ausubel. "The expanded cropland would have come at the expense of other covers, especially forest and grassland."

Using China as an example, Ausubel and his colleagues show that in 2010 China's maize farmers spared 120 million hectares from the land that would have been required with the yields of 1961, twice the area of France. Overall, the researchers found producing an equivalent aggregate of crop production in 2009 required only about 35 percent of the land needed in 1961.
In addition to improved yields achieved by farmers, the researchers credit additional factors leading to peak farmland: parents giving birth to fewer children, and consumers raising their calorie consumption more slowly than their affluence and moderating their meat eating.
"Our analyses over the past 20 years witness food decoupling from land," says Ausubel. "For millennia food production tended to grow in tandem with land used for crops, a fundamental relationship in population and development. Now land for food is flat. If yields had remained at prior levels, immense, continental areas of forest and range and desert would have been shaved and ploughed for human food during the past 50 years. Surprisingly, instead, we find humanity gradually moving toward what we call, with deliberate hyperbole, landless agriculture. We believe humanity now stands at peak farmland, and the 21st century will see release of wide areas of land, hundreds of millions of hectares—more than twice the area of France—for nature."
Provided by Rockefeller University

http://phys.org/news/2012-12-simple-page-chance-website.html

Simple opening page increases chance of website being used further

If a website's homepage is full of information and images then visitors will click away faster than if they first see a quiet and clear page.

So if you want your website to be used, it must have a simple opening page. NWO researcher Rik Crutzen concludes this in a study published online in the scientific journal Interaction Studies.
Psychologist Crutzen investigated the relationship between the complexity of a website and a visitor's willingness to use it. Students in a laboratory were shown various websites about responsible drinking behaviour and they then had to decide very quickly if they were inclined to use the website. The study revealed that the more complex the homepage, the lower the willingness of visitors to remain on the site.

Fancy extras

'Some website builders probably knew this already from experience but now it has also been scientifically demonstrated for the first time: showing a lot of images and information on the homepage puts users off,' says Crutzen explaining his results. 'All those fancy extras give a bad first impression.
The visitor then leaves the page quickly and possibly never returns to the website again.' Quiet opening pages, however, make a good first impression. When the study subjects saw these pages their willingness to view the site further was considerably higher.
Website builders can use the outcomes of Crutzen's research to their advantage. 'They should not immediately overload the visitor with information and images. You do not get a second chance to make a first impression,' says Crutzen. 'This might seem obvious but many websites still have homepages that fail to clearly convey what the website is about.'

Health advice

In a follow-up study Crutzen will focus on the conditions health advice websites must satisfy to be used more. 'We know, for example, that websites with tips and information about giving up smoking, exercising more, and drinking less can help people to live a more healthy life. Unfortunately, however, little use is made of these websites.
A simple opening page is a first possible improvement and with a follow-up study I want to demonstrate what other factors can contribute to a better use of these sites.'
Crutzen is a researcher at Maastricht University. His research is funded by a Veni grant from the Netherlands Organisation for Scientific Research (NWO). *Provided by Netherlands Organisation for Scientific Research (NWO)*

http://bit.ly/VquhbX

2013 Smart Guide: Supercomet to outshine the moon

Doomsayers disappointed by 2012's non-apocalypse will get a sop in 2013 in the form of a rare supercomet.

24 December 2012 by Valerie Jamieson

Once widely seen as a portent of doom, comets are seldom as spectacular as the new arrival, known as C/2012 S1 (ISON), may be. At its peak it may outshine the moon, even by day.
First spotted in September, ISON is rushing towards the sun from the outer solar system. Its closest approach to the sun will be in November, when Timothy Spahr of the Minor Planet Center at Harvard University expects it to put on as good a show as Hale-Bopp did in 1997.

This will be its first trip to the inner solar system, so ISON could contain volatile gases that other comets, making their umpteenth lap around the sun, have lost. That will give us a pristine glimpse of the material in the outer solar system 4.6 billion years ago, when ISON formed.
The year will also herald celestial fireworks of a different flavour, thanks to a gas cloud with three times Earth's mass heading towards the usually placid supermassive black hole at the centre of the galaxy. The collision won't be visible to the naked eye, but X-ray telescopes will pick up radiation from the shock wave created as the cloud slams into the halo of hot gas around the hole.
As the black hole, called Sagittarius A*, sits a mere 25,000 light years away - on our cosmic doorstep - the crash should provide an unprecedented view of material ploughing into a black hole. It could even yield important clues about what happened 300 years ago, when the black hole was much brighter than now.

http://www.eurekalert.org/pub_releases/2012-12/jaaj-elo122012.php

Elevated levels of C-reactive protein appear associated with psychological distress, depression

Elevated levels of C-reactive protein, a marker of inflammatory disease, appear to be associated with increased risk of psychological distress and depression in the general population of adults in Denmark

CHICAGO – Elevated levels of C-reactive protein, a marker of inflammatory disease, appear to be associated with increased risk of psychological distress and depression in the general population of adults in Denmark, according to a report published Online First by Archives of General Psychiatry, a JAMA Network publication.
Depression is one of the leading causes of disability and previous studies suggest that low-grade systemic inflammation may contribute to the development of depression.
C-reactive protein (CRP) is a commonly used marker of inflammation, and inflammatory disease is suspected when CRP levels exceed 10 mg/L.
Researchers are unclear whether and to what extent elevated CRP levels are associated with psychological distress and depression in the general population, according to the study background.
Marie Kim Wium-Andersen, M.D., of Herlev Hospital and Copenhagen University Hospital, Denmark, and colleagues examined whether elevated plasma levels of CRP were associated with distress and depression.
Researchers analyzed CRP levels using data from two general population studies in Copenhagen, which included 73,131 men and women ages 20 to 100 years.
"The main finding of this study consisted of an association of elevated CRP levels with an increased risk for psychological distress and depression in the general population," the authors comment.
Increasing CRP levels were associated with increasing risk for psychological distress and depression in analyses. For self-reported antidepressant use, the odds ratio was 1.38 for CRP levels of 1.01 to 3 mg/L, 2.02 for 3.01 to 10 mg/L, and 2.7 for greater than 10 mg/L compared with 0.01 to 1 mg/L. For prescription of antidepressants, the corresponding odds ratios were 1.08, 1.47 and 1.77, respectively; for hospitalization with depression they were 1.30, 1.84 and 2.27 respectively. Other analyses suggest that increasing CRP levels also were associated with increasing risk for hospitalization with depression, according to the study results.
"More research is needed to establish the direction of the association between CRP and depression because this study and others are primarily cross-sectional. The results also support the initiation of intervention studies to examine whether adding anti-inflammatory drugs to antidepressants for treatment of depression will improve outcome," the authors conclude.
(Arch Gen Psychiatry. Published online December 24, 2012. doi:10.1001/2013.jamapsychiatry.102. Available pre-embargo to the media at http://media.jamanetwork.com.)
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http://www.eurekalert.org/pub_releases/2012-12/ps-fem122012.php

Fluctuating environment may have driven human evolution

A series of rapid environmental changes in East Africa roughly 2 million years ago may be responsible for driving human evolution, according to researchers at Penn State and Rutgers University.

UNIVERSITY PARK, Pa. -- "The landscape early humans were inhabiting transitioned rapidly back and forth between a closed woodland and an open grassland about five to six times during a period of 200,000 years," said Clayton Magill, graduate student in geosciences at Penn State. "These changes happened very abruptly, with each transition occurring over hundreds to just a few thousand years."
According to Katherine Freeman, professor of geosciences, Penn State, the current leading hypothesis suggests that evolutionary changes among humans during the period the team investigated were related to a long, steady environmental change or even one big change in climate.

"There is a view this time in Africa was the 'Great Drying,' when the environment slowly dried out over 3 million years," she said. "But our data show that it was not a grand progression towards dry; the environment was highly variable."
According to Magill, many anthropologists believe that variability of experience can trigger cognitive development.
"Early humans went from having trees available to having only grasses available in just 10 to 100 generations, and their diets would have had to change in response," he said. "Changes in food availability, food type, or the way you get food can trigger evolutionary mechanisms to deal with those changes. The result can be increased brain size and cognition, changes in locomotion and even social changes -- how you interact with others in a group. Our data are consistent with these hypotheses. We show that the environment changed dramatically over a short time, and this variability coincides with an important period in our human evolution when the genus Homo was first established and when there was first evidence of tool use."

The researchers examined lake sediments from Olduvai Gorge in northern Tanzania, looking for biomarkers -- fossil molecules -- from ancient trees and grasses. **Gail Ashley**

The researchers -- including Gail Ashley, professor of earth and planetary sciences, Rutgers University -- examined lake sediments from Olduvai Gorge in northern Tanzania. They removed the organic matter that had either washed or was blown into the lake from the surrounding vegetation, microbes and other organisms 2 million years ago from the sediments. In particular, they looked at biomarkers -- fossil molecules from ancient organisms -- from the waxy coating on plant leaves.
"We looked at leaf waxes because they're tough, they survive well in the sediment," said Freeman.
The team used gas chromatography and mass spectrometry to determine the relative abundances of different leaf waxes and the abundance of carbon isotopes for different leaf waxes. The data enabled them to reconstruct the types of vegetation present in the Olduvai Gorge area at very specific time intervals.
The results showed that the environment transitioned rapidly back and forth between a closed woodland and an open grassland.
To find out what caused this rapid transitioning, the researchers used statistical and mathematical models to correlate the changes they saw in the environment with other things that may have been happening at the time, including changes in the Earth's movement and changes in sea-surface temperatures.
"The orbit of the Earth around the sun slowly changes with time," said Freeman. "These changes were tied to the local climate at Olduvai Gorge through changes in the monsoon system in Africa. Slight changes in the amount of sunshine changed the intensity of atmospheric circulation and the supply of water. The rain patterns that drive the plant patterns follow this monsoon circulation. We found a correlation between changes in the environment and planetary movement."
The team also found a correlation between changes in the environment and sea-surface temperature in the tropics.
"We find complementary forcing mechanisms: one is the way Earth orbits, and the other is variation in ocean temperatures surrounding Africa," Freeman said. The researchers recently published their results in the Proceedings of the National Academy of Sciences along with another paper in the same issue that builds on these findings. The second paper shows that rainfall was greater when there were trees around and less when there was a grassland.
"The research points to the importance of water in an arid landscape like Africa," said Magill. "The plants are so intimately tied to the water that if you have water shortages, they usually lead to food insecurity.
"Together, these two papers shine light on human evolution because we now have an adaptive perspective. We understand, at least to a first approximation, what kinds of conditions were prevalent in that area and we show that changes in food and water were linked to major evolutionary changes."
The National Science Foundation funded this research.