http://www.eurekalert.org/pub_releases/2012-07/niob-vaa070912.php

Vaccine and antibiotics stabilized so refrigeration is not needed -- NIH study

Could pave way for development of enhanced delivery and storage in third world, save billions in cost

Researchers funded by the National Institutes of Health have developed a new silk-based stabilizer that, in the laboratory, kept some vaccines and antibiotics stable up to temperatures of 140 degrees Fahrenheit. This provides a new avenue toward eliminating the need to keep some vaccines and antibiotics refrigerated, which could save billions of dollars every year and increase accessibility to third world populations.

Vaccines and antibiotics often need to be refrigerated to prevent alteration of their chemical structures; such alteration can result in less potent or ineffective medications.

By immobilizing their bioactive molecules using silk protein matrices, researchers were able to protect and stabilize both live vaccines and antibiotics when stored at higher than recommended temperatures for periods far longer than recommended.

The research was led by grantees of NIH's National Institute of Biomedical Imaging and Bioengineering (NIBIB), David Kaplan, Ph.D., and Jeney Zhang, Ph.D. candidate, at Tufts University School of Engineering in Medford, Mass. The National Eye Institute and the National Institute of Dental and Craniofacial Research at NIH also contributed to this research. The researchers reported on their findings in the online issue of Proceedings of the National Academy of Sciences on July 9, 2012.

"This truly exciting development is the culmination of years of creative exploration and research focused on a major problem in the delivery of health care. Dr. Kaplan and his team have done a masterful job at both understanding the key properties of silk, and applying these insights to a global medical challenge," said NIBIB Director Roderic I. Pettigrew, Ph.D., M.D. "This is also a wonderful validation of the type of team science we see in our Biotechnology Resource and Development Centers and their ability to combine cutting edge science in a number of fields to a variety of health needs."

Pettigrew also points out that the next step is to test it in the field.

Keeping medications cold from production until they are used in treatment is a costly process, accounting for as much as 80 percent of the price of vaccinations. The need for a cold chain has been a difficulty for health care providers, aid organizations, scientists and pharmaceutical companies for decades, especially in settings where electricity is limited. Failures in the chain result in the loss of nearly half of all global vaccines, according to researchers.

In an attempt to solve this problem, Kaplan and his lab have been working extensively with silk films that essentially wrap up the live bioactive molecules present in antibiotics and vaccines. This protects these essential bioactive elements, and so can greatly extend the shelf-life of the medication.

Silk is used because it is a protein polymer with a chemistry, structure, and assembly that can generate a unique environment, making it an attractive candidate for the stabilization of bioactive molecules over extended periods of time.

To test their new silk stabilizers, Kaplan's team stored the measles, mumps, and rubella (MMR) vaccines for six months at the recommended 39.2 degrees Fahrenheit, as well as at 77, 98.6 , and 113 degrees Fahrenheit. The results show that encapsulation in the new silk films maintained the potency with minimal loss over time and enhanced stability, even at very high storage temperatures.

Similarly, antibiotics entrapped in silk films maintained near optimal activity even at temperatures as high as 140 degrees. In addition, Kaplan's group found that these silk films had the added benefit of protecting one antibiotic against the detrimental effects of light exposure.

The silk stabilizers are likely to combine well with Kaplan's previously developed silk microneedle system. These tiny needles can deliver medication directly to skin cells that contain a specified antigen. This targeted approach permits administration of lower doses of medication or vaccine and generates longer-lasting immune responses. The combination could prove to be a simple way to stabilize, distribute, and deliver the medication in one system.

Thus, for vaccines and antibiotics, the use of a silk carrier reduces the detrimental effects of heat and humidity.

"New studies are already under way," says Dr. Kaplan. "We have already begun trying to broaden the impact of what we're doing to apply to all vaccines.

Based on what we've seen with other proteins, peptides, and enzymes, there's no reason to believe that this wouldn't be universal. This could potentially eliminate the need for a cold-chain system, greatly decreasing costs and enabling more widespread availability of these life-saving drugs."

http://www.eurekalert.org/pub_releases/2012-07/uota-smm070912.php

Small molecule may play big role in Alzheimer's disease

Simulations by UC Santa Barbara researchers improve understanding of plaque formation and suggest new diagnostic and treatment options

Alzheimer's disease is one of the most dreaded and debilitating illnesses one can develop. Currently, the disease afflicts 6.5 million Americans and the Alzheimer's Association projects it to increase to between 11 and 16 million, or 1 in 85 people, by 2050.

Cell death in the brain causes one to grow forgetful, confused and, eventually, catatonic. Recently approved drugs provide mild relief for symptoms but there is no consensus on the underlying mechanism of the disease.

"We don't know what the problem is in terms of toxicity," said Joan-Emma Shea, professor of chemistry and biochemistry at the University of California, Santa Barbara (UCSB). "This makes the disease difficult to cure."

Accumulations of amyloid plaques have long been associated with the disease and were presumed to be its cause. These long knotty fibrils, formed from misfolded protein fragments, are almost always found in the brains of diseased patients. Because of their ubiquity, amyloid fibrils were considered a potential source of the toxicity that causes cell death in the brain. However, the quantity of fibrils does not correspond with the degree of dementia and other symptoms.

New findings support a hypothesis that fibrils are a by-product of the disease rather than the toxic agent itself. This paradigm shift changes the focus of inquiry to smaller, intermediate molecules that form and dissipate quickly. These molecules are difficult to perceive in brain tissue.

Shea's group uses computer simulations to understand the formation of toxic entities in the brain. Since 2007, Shea has run thousands of simulations of amyloid peptides using the Ranger supercomputer at the Texas Advanced Computing Center (TACC) to better understand the structure, formation and behavior of amyloid accumulations.

"We can identify the important structures or conformations that are adopted by these peptides at a resolution that exceeds what can be done experimentally," she explained. "This helps us understand what structures lead to a self-assembly."

For decades, it was believed that fibrils were a toxic species, but increasingly researchers are looking at small, soluble precursor forms of the fibrils, known as oligomers. "These are difficult to detect experimentally because they tend to be transient species," Shea said "There's no consensus on how big they are. There are still a lot of debates."

Shea and Michael Bowers, professor of chemistry and biochemistry at UCSB and Shea's experimental collaborator, believe the transient oligomers may be responsible for the onset of the disease through interactions with the cell membrane.

"These oligomers may be toxic by inserting themselves into membranes and causing a damage to the membrane," she said. "The membrane is critical for the cell viability."

In 2007, Shea and Bowers received a grant from the National Institutes of Health to investigate this theory. Together, they have spent the last five years looking at small peptide-based inhibitors that would prevent these oligomers from forming.

"If you can prevent the oligomers from forming, you can limit toxicity," Shea said.

In a recent paper currently in press in Biophysical Journal, Shea and postdoctoral researcher Luca Larini studied the conformations adopted by small oligomers of peptide amyloids encountered within the cell. They found that hairpin-shaped forms of the peptide initiated the aggregation of oligomers that ultimately led to the formation of a fibril. Like an old slapstick routine where one person trips, another trips over them, and eventually a pile forms, the misfolded proteins in the brain cells of those with Alzheimer's recruit other misfolded proteins and eventually grow into a large mass.

Shea's simulations have not only helped uncover the possible role of oligomers in the onset of Alzheimer's, but they are aiding in research that is trying to stop oligomer formation in the first place. A paper in the November 2011 edition of Biochemistry, co-authored with the Bowers group, described how a class of small molecules known as c-terminal inhibitors was able to stop the formation of oligomers, possibly halting disease progression before it is too late.

"Dr. Shea's simulations put a molecular face on the cross sections and oligomer distributions that we experimentally measure," said Bowers. " Of significant importance is the simulation of the ABeta42 monomer structure that very nicely correlated with our experiments. Also of importance are calculations on the sites and mechanism of attachment of potential therapeutic agents that we are testing as ABeta aggregation inhibitors."

Simulations on Ranger helped researchers identify where the inhibitors bind and led to new ideas about how inhibition can be improved.
"Dr. Shea is clearly at the top of the large cohort of simulators in her age group," Bowers said.
Through a related investigation, Shea and postdoctoral researcher Chun Wu solved the long-standing mystery of why Thioflavin T, a dye commonly used in brain imaging, is able to bind to amyloid proteins. Her molecular dynamics simulations identified the specific hydrophobic motif in the peptide to which the dye binds. This pinpoint conclusion now allows chemists and neurological experimentalists to create designer forms of the dye that can be used to improve their diagnostic ability. These results were reported in the Biophysical Journal in March 2011.
"Now that we've established where these molecules bind, we can start tweaking the molecule to try to make binders that have a greater affinity for the fibril. That could be something that would be beneficial for medicine as a better imaging agent," she said.
Shea's simulations of peptide interactions, dyes binding to fibrils, and inhibitors stopping the accumulation of amyloids provide great insights to scientists. The projects required more than 13 million hours of compute time on TACC's Ranger and Lonestar supercomputers since 2009.
"The number of atoms is huge - we need a lot of computational resources to simulate them," Shea said. "Nothing that we're doing here is something that we could do on our home clusters. The scale of it is intractable."
Ranger is one of the top 50 most powerful supercomputers in the world, Funded by the National Science Foundation and deployed in 2008, Ranger helps scientists around the country make discoveries by offering free compute time to academic researchers. The system is part of the Extreme Science and Engineering Discovery Environment (XSEDE), the NSF-funded effort to provide cyberinfrastructure and computing power to the nation's scientists.
In February, Ranger will be decommissioned to make way for Stampede, a new supercomputer 20 times more powerful. Such a system will be required to answer further important questions about Alzheimer's disease.
"With growing computational resources and capabilities, we'll be able to look at how these proteins interact with membranes," Shea said. "We're far away from simulating a whole cell, but we can start incorporating additional elements that may turn out to be important."

http://www.eurekalert.org/pub_releases/2012-07/au-son070912.php

Sounds of northern lights are born close to ground

For the first time, researchers at Aalto University in Finland have located where the sounds associated with the northern lights are created.

The auroral sounds that have been described in folktales and by wilderness wanderers are formed about 70 meters above the ground level in the measured case.
Researchers located the sound sources by installing three separate microphones in an observation site where the auroral sounds were recorded. They then compared sounds captured by the microphones and determined the location of the sound source. The aurora borealis was seen at the observation site. The simultaneous measurements of the geomagnetic disturbances, made by the Finnish Meteorological Institute, showed a typical pattern of the northern lights episodes.
"Our research proved that, during the occurrence of the northern lights, people can hear natural auroral sounds related to what they see. In the past, researchers thought that the aurora borealis was too far away for people to hear the sounds it made. This is true. However, our research proves that the source of the sounds that are associated with the aurora borealis we see is likely caused by the same energetic particles from the sun that create the northern lights far away in the sky. These particles or the geomagnetic disturbance produced by them seem to create sound much closer to the ground," said Professor Unto K. Laine from Aalto University.
Details about how the auroral sounds are created are still a mystery. The sounds do not occur regularly when the northern lights are seen.
The recorded, unamplified sounds can be similar to crackles or muffled bangs which last for only a short period of time. Other people who have heard the auroral sounds have described them as distant noise and sputter. Because of these different descriptions, researchers suspect that there are several mechanisms behind the formation of these auroral sounds. These sounds are so soft that one has to listen very carefully to hear them and to distinguish them from the ambient noise.
The Aalto University researcher's study will be published in the proceedings of the 19th International Congress on Sound and Vibration. The congress is held in Vilnius, Lithuania from 8 to 12 July 2012.
video: http://www.youtube.com/watch?v=NRZfKqhs6rM

http://www.eurekalert.org/pub_releases/2012-07/w-wcm070912.php

Wound care meta-review draws firm conclusions from Cochrane published studies

Robust evidence exists for some wound care interventions, but there are still gaps in current knowledge

Robust evidence exists for some wound care interventions, but there are still gaps in current knowledge requiring international consensus and further high-level clinical evidence, according to a paper published online by BJS, the British Journal of Surgery.

Researchers analysed the findings of 44 Cochrane Systematic Reviews (CSRs) published by the Cochrane Wounds and Peripheral Vascular Disease Groups up to June 2011. The reviews covered CSRs on acute wounds and chronic wounds such as venous, pressure, diabetic and arterial ulcers. This enabled them to identify a number of findings that provide strong clinical evidence for treating specific wound issues.

"Acute and chronic wounds pose a substantial problem in different healthcare settings including emergency departments, nursing homes, home care and family doctor practices" says co-author Dr Dirk Ubbink, from the Academic Medical Centre in Amsterdam, The Netherlands. "Because wounds have a considerable impact on patient health, death, daily functioning and quality of life, they deserve high-quality local and systemic treatment.

"Ideally wound treatment decisions should be based on the best available evidence, integrated with patients' concerns and priorities and fine-tuned by the local resources and skills. In reality, however, treatment decisions are generally based on the personal opinions, experiences and preferences of healthcare professionals, which can vary widely. This is partly due to the overwhelming amount of literature available, which often shows conflicting results. "Our meta-review of the CSRs aims to help clinicians make evidence-based decisions by analysing studies of both local and systemic open wound care."

The meta-review covered 13 CSRs on venous ulcers, 12 on acute wounds, seven on pressure ulcers, six on diabetic ulcers, five on arterial ulcers and five on miscellaneous chronic wounds.

Findings were placed into five categories, based on strong evidence of effect/no effect, limited evidence of effect/no effect and no evidence either way.

Strong findings included:

Acute wounds

Using antibiotics to prevent infections after dog bites is ineffective unless the bites are on the hands.

Systemic treatment with therapeutic touch does not have any additional effect on wound healing compared to placebo or non-treatment after minor surgery.

Cleansing pin site wounds using saline, alcohol, hydrogen peroxide or antibacterial soap to prevent infections is no more effective than no cleansing.

Topical honey reduces wound healing time when compared to film or gauze-based dressings for burns.

Silver sulfadiazine should not be used for burns as trials show this can delay wound healing and increase pain and infection rates.

Drinking quality tap water is better for cleansing lacerations and acute soft tissue wounds than sterile saline solutions.

Chronic wounds

Systemic treatment of venous ulcers with pentoxyfylline increases complete wound healing and compression therapy, using high compression, multi-component systems or elastic bandages, is most effective.

Using hyperbaric oxygen therapy decreases major amputations in diabetic ulcers and local hydrogels should be used after debridement to promote wound healing.

Systemic prostanoids should be used to relieve rest pain and improve ulcer healing in patients with critical leg ischaemia and spinal cord stimulation improves limb salvage.

Using high-specification foam mattresses and low air loss mattresses can prevent pressure ulcers on the ward and pressure-relieving overlays are recommended on operation tables. Using local therapeutic ultrasound is not recommended for healing pressure ulcers.

"Our meta-review drew 33 conclusions with strong evidence and 18 conclusions with fairly strong evidence from the CSRs we studied" says lead author Dr Fleur Brölmann. "Evidence was not available or insufficient in the remaining 58."

The paper has been published online early ahead of print publication in the September issue of BJS. It can be viewed free at: http://onlinelibrary.wiley.com/doi/10.1002/bjs.8810/full

A podcast discussion featuring the author and other clinicians can be found at http://www.yada-yada.co.uk/Blackwell/BJS/BJS_wound_dressing.mp3

http://www.eurekalert.org/pub_releases/2012-07/fj-faa070912.php

Frankincense as a medicine

Pharmacists of University Jena clarify the anti-inflammatory impact of boswellic acids

Jena (Germany) It was one of the gifts of the Magi – in addition to myrrh and gold they offered frankincense to the newly born baby Jesus. Since the ancient world the aromatic fragrance of burning Boswellia resin has been part of many religious ceremonies and is still used as a means to indicate special festive atmosphere in the church today. But frankincense can do much more: "The resin from the trunk of Boswellia trees contains anti-inflammatory substances," Professor Dr. Oliver Werz of the Friedrich Schiller University Jena (Germany) says. The chair of Pharmaceutical and Medical Chemistry is convinced that these substances can be very beneficial in therapies against diseases like asthma, rheumatoid arthritis or atopic dermatitis.

However, so far the active substances in frankincense cannot at present be found in drugs in German pharmacies, as the pharmacological impact of frankincense hasn't been thoroughly investigated. "Although Boswellia resin has been used for thousands of years in the Ayurvedic medicine for instance, the clinical studies we have so far are not suffice for a license in Germany and Europe," Professor Werz explains.

But that could change. As part of a mutual project with partners of the University Saarbrücken and a start-up company, Professor Werz and his team examined the curative effect of frankincense. In this project the researchers were able to show where exactly the boswellic acids – which are responsible for the impact of the ingredients of the Boswellia resin – actually interfere in the process of inflammation. "Boswellic acids interact with several different proteins that are part of inflammatory reactions, but most of all with an enzyme which is responsible for the synthesis of prostaglandin E2," Oliver Werz points out. Prostaglandin E2 is one of the mediators of the immune response and plays a decisive role in the process of inflammation, in the development of fever and of pain. "Boswellic acids block this enzyme efficiently and thereby reduce the inflammatory reaction," the Jena pharmacist explains. With this, not only a targeted use in the therapy of inflammatory diseases is conceivable. It can also be expected that boswellic acids have less side effects than today's prevalent anti-inflammatory treatments like diclofenac or indometacin. Their impact is less specific, they can increase the risk of stomach ulcers and can negatively affect renal function.

In their latest study the researchers around Professor Werz additionally compared the resin of different kinds of frankincense in its anti-inflammatory impact. There are more than ten Boswellia species in the world. The most well-known and widely-used one is the Boswellia serrata from Northern and central India. "We were able to show that the resin of the Boswellia papyrifera is ten times more potent," Professor Werz explains a further result of his research. This species mostly occurs in the Northeast of Africa (Ethiopia, Somalia) and on the Arabian Peninsula (Yemen, Oman).

Whether frankincense will become accepted, is indeed not only due to the outcome of the clinical examination which is yet to come. "Boswellic acids exclusively occur in the resin of Boswellia trees and are very difficult to produce synthetically," Werz points out. Therefore these trees are the only source of these promising active ingredients. However Boswellia trees are already an endangered tree species. In many places they are just being used as heating fuel. "Thereby without sustained protection not only plant species are endangered but at the same time medicine loses promising active ingredients," Professor Werz warns.

http://www.sciencedaily.com/releases/2012/07/120709121626.htm

Handlebar Level Can Affect Sexual Health of Female Cyclists

A new study reveals that handlebar position is associated with changes in genital sensation in female cyclists.

ScienceDaily - A new study published in The Journal of Sexual Medicine reveals that handlebar position is associated with changes in genital sensation in female cyclists.

Led by Marsha K. Guess, MD, MS, of Yale University School of Medicine, researchers evaluated bicycle set-up in terms of the relationship between the seat and the handlebars. 48 competitive women cyclists were studied. Researchers measured saddle pressures and sensation in the genital region to see if placing handlebars in different positions affects pressure and sensation in the genital region. Results showed that placing the handlebar lower than the seat was associated with increased pressure on the genital region and decreased sensation (reduced ability to detect vibration).

"Modifying bicycle set-up may help prevent genital nerve damage in female cyclists," Guess notes. "Chronic insult to the genital nerves from increased saddle pressures could potentially result in sexual dysfunction."

"There are a myriad of factors affecting women's sexual function. If women can minimize pressure application to the genital tissues merely by repositioning their handlebars higher, to increase sitting upright, and thereby

maximize pressure application to the woman's sit bones, then they are one step closer to maintaining their very important sexual health," explained Irwin Goldstein, editor-in-chief of The Journal of Sexual Medicine.

Sarah N. Partin, Kathleen A. Connell, Steven Schrader, Julie LaCombe, Brian Lowe, Anne Sweeney, Susan Reutman, Andrea Wang, Christine Toennis, Arnold Melman, Madgy Mikhail, Marsha K. Guess. The Bar Sinister: Does Handlebar Level Damage the Pelvic Floor in Female Cyclists? The Journal of Sexual Medicine, 2012; 9 (5): 1367 DOI: 10.1111/j.1743-6109.2012.02680.x

http://www.wired.com/wiredscience/2012/07/sexual-selection-challenge/

Traditional Sexual Values Challenged in Classic Animal Study

The idea that animal evolution is shaped by males boasting and fighting to win female favor is a central biological dogma.

By Brandon Keim

Females pick males whose exaggerated traits suggest virility, thus producing peacock feathers and sage grouse struts. Males compete for female favor, hence a stag's antlers and fights for territorial domination. These are the main engines of sexual selection, the default explanation for differences between the sexes.

Under closer scrutiny, however, the dogma doesn't seem to hold. A new replication of English geneticist Angust Bateman's foundational mid-20th century mate-choice study, a study that reinforced sexual selection assumptions and shaped decades of research, came to very different conclusions than the original.

Bateman's refutation may be an exclamation point for critics who say the evolutionary dance between sexes is far richer and more complicated than a male-dominated two-step.

"Our expectations have been so strongly affected by Bateman," said evolutionary biologist Patricia Gowaty of the University of California, Los Angeles, who led the study's replication, published in June in Proceedings of the National Academy of Sciences. "It's almost as though our imaginations were limited."

The original study, published in 1948, followed on a line of thinking that originated with Charles Darwin, who saw in the fantastically colorful feathers of male peafowl a trait that made no evolutionary sense except as an advertisement to prospective mates. Peacocks and peahens had resembled each other long ago, he surmised, but hens learned that bright, spotted feathers signified fitness. Magnified over evolutionary time, their preference produced the peacocks' otherwise impractical plumage.

For Darwin, this demonstrated a universal principle: Males benefit by mating frequently and indiscriminately, with each successful copulation representing an extra chance to pass on their traits, while females mate infrequently, invest energy in rearing offspring and generally benefit by being choosy about mates. From that dynamic would emerge physical and behavioral differences between sexes.

Bateman tested the principle using what were, for his time, cutting-edge genetic tools. He bred fruit flies that each contained a single pronounced trait, such as tiny heads or shriveled wings or slit-shaped eyes. By looking for these traits in offspring, it was possible to identify their parents.

Tests affirmed Darwin's ideas and were codified into what became known as Bateman's principles, which mathematically described how between-sex differences in mating benefits and reproductive fitness could be measured. The paper has been cited nearly 2,000 times and its ideas became assumptions with cultural resonance. "The word excess has no meaning for a male," wrote Richard Dawkins in The Selfish Gene.

Over time, however, rumblings of discontent grew. A few biologists, Gowaty among them, suggested that mating patterns measured by Bateman could have been explained by chance or environment. Yet nobody put Bateman's study to the ultimate test - until now.

In the replication, Gowaty and geneticists Wyatt Anderson and Yong-Kyu Kim of the University of Georgia duplicated Bateman's study design, even breeding fresh generations of fruit flies with the same exaggerated physical traits. They noticed something that Bateman didn't consider important: Flies with pronounced traits from two parents, such as shriveled wings and tiny heads, were unfit and died before hatching.

Bateman only counted adult flies, but Gowaty and Anderson included the deceased juveniles. Their results show that Bateman's approach was skewed, causing him to overestimate the number of adults that hadn't mated at all, underestimate the number who mated multiple times, and to under-count mothers. In short, there was no evidence for indiscriminately mating males and finicky, seldom-mating females.

"Bateman's ideas … have helped to define what we mean by sexual selection and how best to measure it," wrote Zuleyma Tang-Martínez, a biologist at the University of Missouri-St. Louis, in a separate commentary in PNAS. She said the new results are "likely to lead to a paradigm shift in the study of sexual selection and related topics."

A contrary interpretation was offered by evolutionary biologist Adam Jones of the University of Texas. "This study, while interesting, is not paradigm challenging," he said. "We now have studies from many other systems

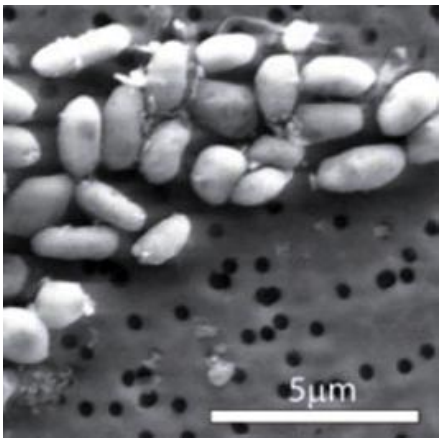
other than Drosophila (fruit flies) confirming the main conclusions of Bateman's original study. Thus, even if Bateman's study was flawed, as it certainly was by today's standards, the impact of his study on the field of evolutionary biology is secure."
Among aracaris, a relative of the toucan, males and females possess equally exaggerated, colorful features. Traditional ideas of sexual selection don't seem to explain why. Photo: lowjumpingfrog/Flickr
Jones' own research has demonstrated that classic, Bateman-style sexual selection does explain sexual differences in animals like the rough-skinned newt, and it unquestionably applies to many species.
But Martinez and Gowaty say that traditional views on sexual selection have skewed science. "Bateman's ideas and conclusions … have helped to define what we mean by sexual selection," wrote Martinez. "It is fairly common for studies of Bateman gradients to disregard alternative explanations."
Gowaty said her work in the 1980s on female bluebirds, who mate frequently with multiple males, was originally greeted with incredulity. "I had colleagues say that it couldn't happen, that it was rape," she said. Since then, research has painted a picture of animal reproduction more colorful than Darwin or Bateman ever surmised. Male competition and female choosiness only sometimes describe the variety of reproductive habits influencing animal evolution.
Gowaty hopes that revisiting Bateman's study will encourage people to see "alternative" mating strategies as unexceptional, though she said the fundamental implication is less about animal behavior than the importance of challenging received wisdom. "We believed the results so thoroughly, it didn't occur to people to replicate the study," Gowaty said.
"I wonder if we shouldn't all be a little more self-skeptical," she continued. "If we missed for so long that Bateman was inadequate to his task, what might we be missing in more modern studies?"
Citations: "No evidence of sexual selection in a repetition of Bateman's classic study of Drosophila melanogaster." By Patricia Adair Gowaty, Yong-Kyu Kim and Wyatt W. Anderson. Proceedings of the National Academy of Sciences, June 11, 2012.
"Replication of Bateman challenges the paradigm." By Zuleyma Tang-Martínez. Proceedings of the National Academy of Sciences, July 3, 2012.

http://www.scientificamerican.com/article.cfm?id=arsenic-tolerant-bacterium-needs-phosphorus

Notorious Arsenic-Tolerant Bacterium Needs Phosphorus After All

Two teams have repeated a much-debated study and found that the chemical rules of life remain unbroken

By Quirin Schiermeier and Nature magazine | Monday, July 9, 2012 | 3



GFAJ-1 bacteria can live in high concentrations of arsenic - but do not incorporate the element in to their DNA. Image: Science/AAAS

Nature - After 18 months of controversy, the official verdict is in: an arsenic-tolerant bacterium found in California's Mono Lake cannot live without phosphorus.
In 2010, a group led by Felisa Wolfe-Simon, a microbiologist now at the Lawrence Berkeley National Laboratory in Berkeley, California, reported online in Science1 that the Halomonadaceae bacterium GFAJ-1 could include atoms of arsenic instead of phosphorus in crucial biochemicals such as DNA.
The bacteria were discovered thriving in the arsenic-rich sediment of the shallow saline Mono Lake, famed for its appearance on a picture-postcard insert to Pink Floyd's 1975 album Wish You Were Here.
All known forms of life depend on at least six elements: hydrogen, carbon, nitrogen, oxygen, phosphorus and sulphur. Arsenic has some chemical similarities with phosphorus, but is usually toxic to life, so the suggestion that it could sustain life triggered a storm of questions, as well as criticism about how the find was revealed at an enthusiastic NASA press conference (see 'Microbe gets toxic response').
As a result of the controversy, when Wolfe-Simon's paper appeared in print in Science last June, it was accompanied by eight technical comments2–9 from scientists responding to it.
Rosie Redfield, a microbiologist at the University of British Columbia in Vancouver, Canada, set about testing the finding. Earlier this year, she said that she could not reproduce Wolfe-Simon's results in laboratory experiments (see 'Study challenges existence of arsenic-based life').
Redfield is now a co-author of one of two papers that confirm that, although the bacteria can tolerate arsenic, they do depend on phosphorus. The papers were published10, 11 by Science on 8 July.

Toxicity tolerance

Redfield and her colleagues report10 that when GFAJ-1 bacteria were grown in a medium containing arsenic and a very small amount of phosphorus, their DNA contained no detectable arsenic compounds, such as arsenate (the arsenic analogue of phosphate). In the second paper, Julia Vorholt, a microbiologist at the Federal Institute of Technology in Zurich, Switzerland, and her colleagues report11 that the bacterium cannot grow in a phosphorus-free medium in the presence of arsenate. It can, however, grow in low-phosphate conditions in the presence of arsenate. GFAJ-1 "is an arsenate-resistant, but still a phosphate-dependent bacterium", the team writes.

"I think we have now very solid evidence that the metabolism of GFAJ-1 is as dependent on phosphorus, as are all other known forms of organic life," says Vorholt. "These very robust and very well-adapted microbes appear to be able to efficiently extract nutrients from their extremely phosphorus-poor environments."

The samples that Wolfe-Simon's team had used for their original experiments apparently contained larger concentrations of phosphorus than was first thought, Vorholt adds.

In a statement, Science said: "The new research shows that GFAJ-1 does not break the long-held rules of life, contrary to how Wolfe-Simon had interpreted her group's data."

"The original GFAJ-1 paper emphasized tolerance to arsenic, but suggested the cells required phosphorus, as seen in these two new papers," says Wolfe-Simon. "However, our data implied that a very small amount of arsenate may be incorporated into cells and biomolecules, helping cells to survive in environments of high arsenate and very low phosphate. Such low amounts of arsenic incorporation may be challenging to find and unstable once cells are opened."

The story of GFAJ-1 is far from over, she adds. "The key questions are: how do these cells thrive in lethal concentrations of arsenic? And where does the arsenic go?"

http://www.sciencedaily.com/releases/2012/07/120709155425.htm

Drug from Mediterranean Weed Kills Tumor Cells in Mice

Scientists have developed a novel anticancer drug from a weedlike plant that has been shown to destroy cancers, acting like a "molecular grenade

ScienceDaily - Scientists at the Johns Hopkins Kimmel Cancer Center, working with Danish researchers, have developed a novel anticancer drug designed to travel -- undetected by normal cells -- through the bloodstream until activated by specific cancer proteins. The drug, made from a weedlike plant, has been shown to destroy cancers and their direct blood supplies, acting like a "molecular grenade," and sparing healthy blood vessels and tissues.

In laboratory studies, researchers said they found that a three-day course of the drug, called G202, reduced the size of human prostate tumors grown in mice by an average of 50 percent within 30 days. In a direct comparison, G202 outperformed the chemotherapy drug docetaxel, reducing seven of nine human prostate tumors in mice by more than 50 percent in 21 days. Docetaxel reduced one of eight human prostate tumors in mice by more than 50 percent in the same time period.

In a report June 27 in the journal Science Translational Medicine, the researchers also reported that G202 produced at least 50 percent regression in models of human breast cancer, kidney cancer and bladder cancer. Based on these results, Johns Hopkins physicians have performed a phase I clinical trial to assess safety of the drug and have thus far treated 29 patients with advanced cancer. In addition to Johns Hopkins, the University of Wisconsin and the University of Texas-San Antonio are participating in the trial. A phase II trial to test the drug in patients with prostate cancer and liver cancer is planned.

The drug G202 is chemically derived from a weed called Thapsia garganica that grows naturally in the Mediterranean region. The plant makes a product, dubbed thapsigargin, that since the time of ancient Greece has been known to be toxic to animals. In Arab caravans, the plant was known as the "death carrot" because it would kill camels if they ate it, the researchers noted.

"Our goal was to try to re-engineer this very toxic natural plant product into a drug we might use to treat human cancer," says lead study author Samuel Denmeade, M.D., professor of oncology, urology, pharmacology and molecular sciences. "We achieved this by creating a format that requires modification by cells to release the active drug."

By disassembling thapsigargin and chemically modifying it, the researchers created a form that Denmeade likens to a hand grenade with an intact pin. The drug can be injected and can travel through the bloodstream until it finds the site of cancer cells and hits a protein called prostate-specific membrane antigen (PSMA). PSMA is released by cells lining tumors of the prostate and other areas, and in effect "pulls the pin" on G202, releasing cell-killing agents into the tumor and the blood vessels that feed it, as well as to other cells in the

vicinity. Specifically, G202 blocks the function of a protein called the SERCA pump, a housekeeping protein necessary for cell survival that keeps the level of calcium in the cell at the correct level, the researchers report. "The exciting thing is that the cancer itself is activating its own demise," says senior study author John Isaacs, Ph.D., professor of oncology, urology, chemical and biomedical engineering at Johns Hopkins.

Because the drug is targeted to the SERCA pump, which all cells need to stay alive, researchers say it will be difficult for tumor cells to become resistant to the drug, because they cannot stop making the protein.

S. R. Denmeade, A. M. Mhaka, D. M. Rosen, W. N. Brennen, S. Dalrymple, I. Dach, C. Olesen, B. Gurel, A. M. DeMarzo, G. Wilding, M. A. Carducci, C. A. Dionne, J. V. Moller, P. Nissen, S. B. Christensen, J. T. Isaacs. Engineering a Prostate-Specific Membrane Antigen-Activated Tumor Endothelial Cell Prodrug for Cancer Therapy. Science Translational Medicine, 2012; 4 (140): 140ra86 DOI: 10.1126/scitranslmed.3003886

http://www.scientificamerican.com/article.cfm?id=single-genetic-variant-linked-multiple-sclerosis-risk

Single Genetic Variant Is Linked to Multiple Sclerosis Risk

The discovery could help to improve clinical trials of potential therapies

By Ewen Callaway and Nature magazine | July 9, 2012 | 1

Like diabetes, most forms of cancer and other common diseases, there is no single gene that causes the autoimmune condition multiple sclerosis (MS). Dozens of genetic variations act in concert with environmental factors to cause the debilitating neurological disease. Yet a single genetic variant may explain why drugs that treat other autoimmune diseases tend to make MS symptoms worse, and could identify other MS patients who might benefit from the therapies. Researchers say that the findings, which are published online in Nature, also highlight how genome-wide association studies (GWAS) can yield useful medical insights.

GWAS compare thousands of people who have a particular disease, detailing hundreds of thousands of genetic variations between them. The goal is to identify variations that are more common in people with the condition than in healthy people. Most such studies uncover scores of genetic variants associated with the disease in question, each increasing a person's chances of developing the condition by a small percentage.

Such is the case for a DNA letter in the gene that encodes the protein called tumour necrosis factor receptor 1 (TNFR1). The protein senses a potent immune molecule called tumour necrosis factor (TNF) that destroys cancerous cells but that is also implicated in autoimmune disease. People of European ancestry who have two 'A's at that particular spot on the genome are 12% more likely to develop MS than those with two 'G's at that spot.

Lars Fugger, a neurologist at the University of Oxford, UK, and his team discovered that the variant A shortens the TNF receptor 1 protein. Normally, the protein sits within a cell's membrane, where it can sense TNF molecules circulating outside the cell and convey their instructions to that cell. The protein starts a cascade that can lead to inflammation and cell death. But the shortened form of the protein never makes it into the membrane, Fugger's team found. Instead it is probably shunted outside the cell, where it can bind TNF molecules, stopping them from signalling to cells.

Biologic drugs that block TNF from signalling to cells have revolutionized treatment for autoimmune conditions such as rheumatoid arthritis over the past decade, says Fugger. Yet despite achieving some encouraging results in animal models of MS, drugs that block the activity of TNF tend not work in patients with MS. In fact, they usually make symptoms worse, and they may even have caused the disease in people predisposed to it, says Fugger. He thinks the variation his team studied could explain why.

Fugger says it is unclear how TNF influences MS, but in patients with the gene variant, the TNF-blocking drugs could be providing a double-whammy by suppressing TNF signalling further. The normal receptor prevents mice from developing symptoms of a model of MS. It is not clear whether TNF-blocking drugs might help MS patients who don't have the TNF variant, says Fugger, but taking a closer look at the genetics of the patients who received the treatment as part of clinical trials in the 1990s could provide the answer.

Alastair Compston, a neurologist at the University of Cambridge, UK, who led a consortium that last year identified dozens of genetic risk factors for MS in nearly 10,000 MS patients, says there is overwhelming genetic evidence that TNF is somehow involved in the condition. "Therefore the potential for manipulating it therapeutically is real," he says.

Fugger hopes that his team's study will also help to dispel the notion that genome-wide association studies will never offer much that can be used in patient care (see 'Human genetics: Hit or miss'). If doctors had known that TNF-blocking drugs mimic the effects of a major risk factor for the disease, they might have designed their clinical trials differently, he says. "The idea is you can use GWAS studies to decide which drugs should be used and which should not be used," Fugger says.

http://boingboing.net/2012/07/09/you-cannot-poison-an-opossum.html

You cannot poison an opossum

As we established last week, biology is freaking crazy.

By Maggie Koerth-Baker at 9:09 am Monday, Jul 9

Here's more proof. For this, you don't even need to go to exotic Australia. The common American opossum produces a protein called Lethal Toxin-Neutralizing Factor (LTNF). This protein does pretty much what the name implies - seeking out potentially deadly poisons and neutralizing them. The benefit: Opossums are all-but immune to the venom of poisonous snakes. (Including the venom of snakes native to continents where the common American opossum does not live.) But it gets weirder, as Jason Bittel explains on the BittelMeThis blog:

So they took some rats and injected them with LTNF, then pumped them full of otherwise lethal doses of venom from Thailand cobras, Australian taipans, Brazilian rattlesnakes, scorpions and honeybees. But the rats just laughed in their faces.

"Dude," said one scientist, "we have to kill these rats. Do you watch AMC's Breaking Bad?" The other scientists nodded of course because everybody watches Breaking Bad. So next they tried to kill the rats with ricin, an extremely lethal poison made from castor beans. (How lethal? Just ask Georgi Markov, the real-life Bulgarian defector killed by a ricin umbrella gun. That's right, I said ricin umbrella gun.)

Alas, the ricin was a no-go. The now-snooty rats danced Ring Around the Rosie.

"That's it!" screeched the lead scientist. "It's time to release the botulinum toxin. Surely this will conquer the awkward opossum's super serum!" But after many maniacal laughs and a few bolts of lightning, the rats were still alive.

(The paper does not mention what became of the super rats. I can only assume they went on to write "The Secret of Nimh" while the evil scientists lost their rat-killing grant.)

Read the research paper

Read the rest of Jason Bittel's story on the strange and wonderful biology of opossums.

http://www.eurekalert.org/pub_releases/2012-07/uoma-uar071012.php

UMass Amherst researchers unravel secrets of parasites' replication

Microbiologists at the University of Massachusetts Amherst have made an advance that could 1 day lead to a new weapon for fighting parasitic diseases such as African sleeping sickness, chagas disease and leishmaniasis

AMHERST, Mass. – A group of diseases that kill millions of people each year can't be touched by antibiotics, and some treatment is so harsh the patient can't survive it. They're caused by parasites, and for decades researchers have searched for a "magic bullet" to kill them without harming the patient. Now, a team of microbiologists at the University of Massachusetts Amherst has made an advance that could one day lead to a new weapon for fighting parasitic diseases such as African sleeping sickness, chagas disease and leishmaniasis.

In the cover article of the current issue of Eukaryotic Cell, parasitologists Michele Klingbeil, doctoral candidate Jeniffer Concepción-Acevedo and colleagues report the first detailed characterization of the way key proteins in the model parasite Trypanosoma brucei organize to replicate its mitochondrial DNA (mtDNA). Understanding this spatial and temporal coordination could mean a foot in the door to launch new attacks on one of the parasites' essential cell processes, Klingbeil says.

She adds, "Parasites such as T. brucei, which causes African sleeping sickness, are not straightforward to treat because they're too much like our own cells. Antibiotics are ineffective, so we treat them as invaders, with toxic chemicals. We are trying to find their weaknesses so we can exploit those and eventually develop a very selective, effective and acceptable treatment."

Advances have not come easily, in part because these parasites have the most complex mitochondrial genome structure in nature, say Klingbeil and Concepción-Acevedo, the lead researcher on the project. To tackle it, they've focused on the trypanosome parasites' extremely complex method of mtDNA replication, which involves kinetoplast DNA or kDNA. Its core components are very unlike DNA replication in animals and human hosts, Klingbeil says, "so if we can inhibit the replication process and take away the kDNA, the parasites will die. That's one way we might be able to kill them."

Trypanosomes' kDNA is found as a nucleoid in the mitochondrion, where it holds many copies of catenated or networked minicircles and maxicircles that look like medieval chain mail under the microscope. These molecules pass information on to daughter cells via DNA polymerases whose job it is to copy all circles in the network. Trypanosomes have six mtDNA polymerases, while humans have just one.

To figure out how these trypanosomal polymerases know when to initiate DNA replication, Concepción-Acevedo set up immunofluorescence experiments focused on tracking a particular one, known as mtDNA polymerase ID (POLID). By fluorescent labeling the POLID protein and tracking it over space and time, Concepción-Acevedo quantified it and clarified its relationship to the overall replication process for the first time in a very discrete time window. The approach immediately paid off.

Klingbeil says, "As soon as Jeny began looking more closely at POLID localization she discovered a novel mechanism for how this protein participates in kDNA replication." In response to kDNA changes during the replication cycle, POLID was dynamically redistributing, or changing location, from the mitochondrial matrix to concentrated foci around the kDNA, and co-localizing with replicating kDNA molecules.

"This had been hypothesized, but never seen before," Klingbeil explains. It was amazing to witness. We visualized a mitochondrial replication protein undergoing dynamic localization for the first time, and linked it to DNA synthesis. No one had ever been able to do that in any mitochondrial DNA replication system before."

This important discovery explains how POLID engages in kDNA replication and opens up new avenues to study and intervene in mitochondrial protein dynamics, say the two parasitologists. Their ultimate success would be to find a chemical to inhibit POLID from carrying out its role during replication and target all parasites with kDNA structures.

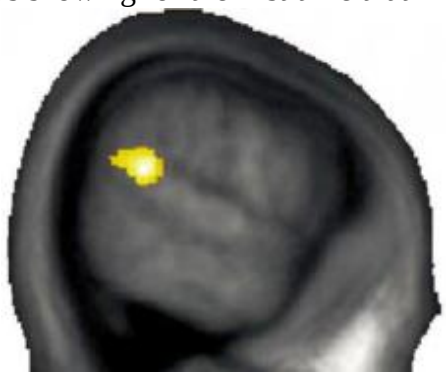
This work was funded by the National Institutes of Health's National Institute of Allergy and Infectious Diseases. Support for Concepción-Acevedo also came from NSF's Northeast Alliance for Graduate Education and the Professoriate program.

http://www.eurekalert.org/pub_releases/2012-07/uoz-tmg071112.php

The more gray matter you have, the more altruistic you are

The volume of a small brain region influences one's predisposition for altruistic behavior.

Researchers from the University of Zurich show that people who behave more altruistically than others have more gray matter at the junction between the parietal and temporal lobe, thus showing for the first time that there is a connection between brain anatomy, brain activity and altruistic behavior.



The junction (yellow) between the parietal and the temporal lobes, in which the relative proportion of gray matter is significantly positively correlated with the propensity for altruistic behavior. **University of Zurich**

Why are some people very selfish and others very altruistic? Previous studies indicated that social categories like gender, income or education can hardly explain differences in altruistic behavior. Recent neuroscience studies have demonstrated that differences in brain structure might be linked to differences in personality traits and abilities. Now, for the first time, a team of researchers from the University of Zurich headed by Ernst Fehr, Director of the Department of Economics, show that there is a connection between brain anatomy and altruistic behavior.

To investigate whether differences in altruistic behavior have neurobiological causes, volunteers were to divide money between themselves and an anonymous other person. The participants always had the option of sacrificing a certain portion of the money for the benefit of the other person. Such a sacrifice can be deemed altruistic because it helps someone else at one's own expense. The researchers found major differences in this respect: Some participants were almost never willing to sacrifice money to benefit others while others behaved very altruistically.

More gray matter

The aim of the study, however, was to find out why there are such differences. Previous studies had shown that a certain region of the brain – the place where the parietal and temporal lobes meet – is linked to the ability to put oneself in someone else's shoes in order to understand their thoughts and feelings. Altruism is probably closely related to this ability. Consequently, the researchers suspected that individual differences in this part of the brain might be linked to differences in altruistic behavior. And, according to Yosuke Morishima, a postdoctoral researcher at the Department of Economics at the University of Zurich, they were right: "People who behaved more altruistically also had a higher proportion of gray matter at the junction between the parietal and temporal lobes."

Differences in brain activity

The participants in the study also displayed marked differences in brain activity while they were deciding how to split up the money. In the case of selfish people, the small brain region behind the ear is already active when the cost of altruistic behavior is very low. In altruistic people, however, this brain region only becomes more active when the cost is very high. The brain region is thus activated especially strongly when people reach the

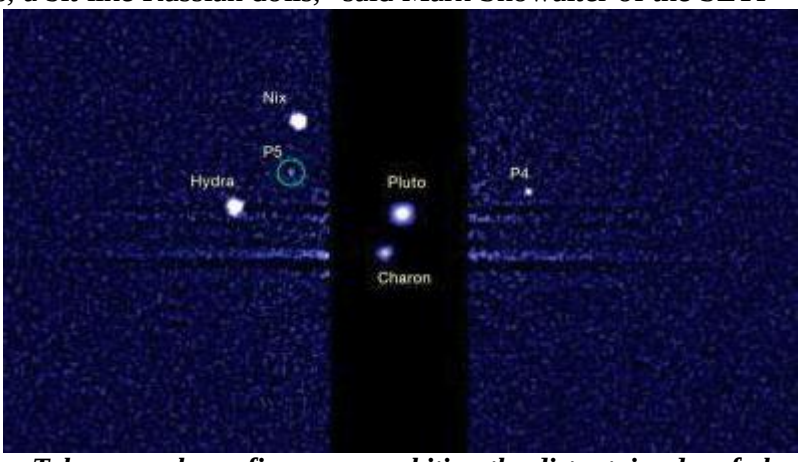
limits of their willingness to behave altruistically. The reason, the researchers suspect, is that this is when there is the greatest need to overcome man's natural self-centeredness by activating this brain region.
Ernst Fehr adds: "These are exciting results for us. However, one should not jump to the conclusion that altruistic behavior is determined by biological factors alone." The volume of gray matter is also influenced by social processes. According to Fehr, the findings therefore raise the fascinating question as to whether it is possible to promote the development of brain regions that are important for altruistic behavior through appropriate training or social norms.
The study is a part of the larger research program "Neurochoice", a project initiated and financed in part by SystemsX.ch

http://www.eurekalert.org/pub_releases/2012-07/eic-hdn071112.php

Hubble discovers new Pluto moon

Pluto's new-found moon, visible as a speck of light in Hubble images, is estimated to be irregular in shape and between 10 and 25 kilometres across.

It is in a 95 000 kilometre-diameter circular orbit around Pluto that is assumed to lie in the same plane as Pluto's other known moons.
"The moons form a series of neatly nested orbits, a bit like Russian dolls," said Mark Showalter of the SETI Institute in Mountain View, USA, leader of the scientific team that discovered the new moon.
The Pluto team is intrigued that such a small planet can have such a complex collection of satellites.
The new discovery provides additional clues for unraveling how the Pluto system formed and evolved.
The favoured theory is that all the moons are relics of a collision between Pluto and another large Kuiper belt [1] object billions of years ago.



This image, taken by the NASA/ESA Hubble Space Telescope, shows five moons orbiting the distant, icy dwarf planet Pluto. The green circle marks the newly discovered moon, designated S/2012 (134340) 1, or P5, as photographed by Hubble's Wide Field Camera 3 on July 7, 2012. Other observations that collectively show the moon's orbital motion were taken on June 26, 27 and 29 and on July 9. The moon is estimated to be 10 to 25 kilometers across. It is in a 95,000 kilometer diameter circular orbit around Pluto that is assumed to be aligned in the same plane as the other satellites in the system. The darker stripe in the center of the image is because the picture is constructed from a long exposure designed to capture the comparatively faint satellites of Nix, Hydra, P4 and S/2012 (134340) 1, and a shorter exposure to capture Pluto and Charon, which are much brighter.
NASA, ESA, and M. Showalter (SETI Institute)

Pluto's largest moon, Charon, was discovered in 1978. Hubble observations in 2006 uncovered two additional small moons, Nix and Hydra. In 2011 another moon, known as P4, was found in Hubble data.
Provisionally designated S/2012 (134340) 1, or P5, the latest moon was detected in nine separate sets of images taken by Hubble's Wide Field Camera 3 on 26, 27 and 29 June, and 7 and 9 July 2012.
New Horizons, a NASA space probe, is currently en route to Pluto, with a high-speed flyby scheduled for 2015. It will return the first ever detailed images of the Pluto system, which is so small and distant that even Hubble can barely see the largest features on its surface.
In the years following the New Horizons Pluto flyby, astronomers plan to use the infrared vision of Hubble's planned successor, the NASA/ESA/CSA James Webb Space Telescope, for follow-up observations.
The James Webb Space Telescope will be able to study the surface chemistry of Pluto, its moons, and many other bodies that lie in the distant Kuiper Belt along with Pluto.
The Hubble Space Telescope is a project of international cooperation between ESA and NASA.
The Pluto team members are M. Showalter (SETI Institute, Mountain View, USA), H.A. Weaver (Applied Physics Laboratory, Johns Hopkins University, Baltimore, USA), and S.A. Stern, A.J. Steffl, and M.W. Buie (Southwest Research Institute, San Antonio, USA).
[1] The Kuiper belt is a region of space in the outer region of the Solar System which contains many small icy objects, and a number of dwarf planets including Pluto.
Image credit: NASA, ESA, and M. Showalter (SETI Institute)

http://www.eurekalert.org/pub_releases/2012-07/ucl-nap070912.php

Native American populations descend from 3 key migrations

Scientists have found that Native American populations arose from at least three migrations

Scientists have found that Native American populations - from Canada to the southern tip of Chile - arose from at least three migrations, with the majority descended entirely from a single group of First American migrants that crossed over through Beringia, a land bridge between Asia and America that existed during the ice ages, more than 15,000 years ago.

By studying variations in Native American DNA sequences, the international team found that while most of the Native American populations arose from the first migration, two subsequent migrations also made important genetic contributions. The paper is published in the journal Nature today.

"For years it has been contentious whether the settlement of the Americas occurred by means of a single or multiple migrations from Siberia," said Professor Andres Ruiz-Linares (UCL Genetics, Evolution and Environment), who coordinated the study. "But our research settles this debate: Native Americans do not stem from a single migration. Our study also begins to cast light on patterns of human dispersal within the Americas."

In the most comprehensive survey of genetic diversity in Native Americans so far, the team took data from 52 Native American and 17 Siberian groups, studying more than 300,000 specific DNA sequence variations called Single Nucleotide Polymorphisms to examine patterns of genetic similarities and differences between the population groups.

The second and third migrations have left an impact only in Arctic populations that speak Eskimo-Aleut languages and in the Canadian Chipewyan who speak a Na-Dene language. However, even these populations have inherited most of their genome from the First American migration. Eskimo-Aleut speakers derive more than 50% of their DNA from First Americans, and the Chipewyan around 90%. This reflects the fact that these two later streams of Asian migration mixed with the First Americans they encountered after they arrived in North America.

"There are at least three deep lineages in Native American populations," said co-author David Reich, Professor of genetics at Harvard Medical School. "The Asian lineage leading to First Americans is the most anciently diverged, whereas the Asian lineages that contributed some of the DNA to Eskimo–Aleut speakers and the Na-Dene-speaking Chipewyan from Canada are more closely related to present-day East Asian populations."

The team also found that once in the Americas, people expanded southward along a route that hugged the coast with populations splitting off along the way. After divergence, there was little gene flow among Native American groups, especially in South America.

Two striking exceptions to this simple dispersal were also discovered. First, Central American Chibchan-speakers have ancestry from both North and South America, reflecting back-migration from South America and mixture of two widely separated strands of Native ancestry. Second, the Naukan and coastal Chukchi from north-eastern Siberia carry 'First American' DNA. Thus, Eskimo-Aleut speakers migrated back to Asia, bringing Native American genes.

The team's analysis was complicated by the influx into the hemisphere of European and African immigrants since 1492 and the 500 years of genetic mixing that followed. To address this, the authors developed methods that allowed them to focus on the sections of peoples' genomes that were of entirely Native American origin.

"The study of Native American populations is technically very challenging because of the widespread occurrence of European and African mixture in Native American groups," said Professor Ruiz-Linares.

"We developed a method to peel back this mixture to learn about the relationships among Native Americans before Europeans and Africans arrived," Professor Reich said, "allowing us to study the history of many more Native American populations than we could have done otherwise."

The assembly of DNA samples from such a diverse range of populations was only possible through a collaboration of an international team of 64 researchers from the Americas (Argentina, Bolivia, Brazil, Canada, Chile, Colombia, Costa Rica, Guatemala, Mexico, Peru, Russia and the USA), Europe (England, France, Spain and Switzerland) and Russia.

1. For more information, please contact Andres Ruiz-Linares on office number +44 (0)20 3108 4003, mobile +44 07900181839, e-mail a.ruizlin@ucl.ac.uk, or David Reich on office number +1 (617) 432-6548, e-mail: reich@genetics.med.harvard.edu.

2. Alternatively, please contact George Wigmore in the UCL Media Relations Office on tel: +44 (0)20 76799041, out of hours +44 (0)7917 271 364, e-mail: g.wigmore@ucl.ac.uk.

3. 'Reconstructing Native American population history' is published in the journal Nature on 1800 London time / 1300 US Eastern Time on 11 July 2012. Journalists can obtain copies of the paper by contacting UCL Media Relations.

http://www.eurekalert.org/pub_releases/2012-07/osu-nss070512.php

New study suggests moderate alcohol consumption may help prevent bone loss

Drinking a moderate amount of alcohol as part of a healthy lifestyle may benefit women's bone health, lowering their risk of developing osteoporosis.

CORVALLIS, Ore. – A new study assessed the effects of alcohol withdrawal on bone turnover in postmenopausal women who drank one or two drinks per day several times a week. Researchers at Oregon State University measured a significant increase in blood markers of bone turnover in women after they stopped drinking for just two weeks.

Bones are in a constant state of remodeling with old bone being removed and replaced. In people with osteoporosis, more bone is lost than reformed resulting in porous, weak bones. About 80 percent of all people with osteoporosis are women, and postmenopausal women face an even greater risk because estrogen, a hormone that helps keep bone remodeling in balance, decreases after menopause.

Past studies have shown that moderate drinkers have a higher bone density than non-drinkers or heavy drinkers, but these studies have provided no explanation for the differences in bone density. Alcohol appears to behave similarly to estrogen in that it reduces bone turnover, the researchers said.

In the current study, published online July 11 in the journal Menopause, researchers in OSU's Skeletal Biology Laboratory studied 40 early postmenopausal women who regularly had one or two drinks a day, were not on any hormone replacement therapies, and had no history of osteoporosis-related fractures.

The researchers found evidence for increased bone turnover – a risk factor for osteoporotic fractures – during the two week period when the participants stopped drinking. Even more surprising: the researchers found that less than a day after the women resumed their normal drinking, their bone turnover rates returned to previous levels.

"Drinking moderately as part of a healthy lifestyle that includes a good diet and exercise may be beneficial for bone health, especially in postmenopausal women," said Urszula Iwaniec, associate professor in the College of Public Health and Human Sciences at OSU and one of the study's authors. "After less than 24 hours to see such a measurable effect was really unexpected."

Iwaniec, OSU's Skeletal Biology Laboratory director Russell Turner, and researcher Gianni Maddalozzo assisted OSU alumna Jill Marrone with the study, which was Marrone's master's thesis.

This study is important because it suggests a cellular mechanism for the increased bone density often observed in postmenopausal women who are moderate drinkers, Turner said.

The researchers said many of the medications to help prevent bone loss are not only expensive, but can have unwanted side effects. While excessive drinking has a negative impact on health, drinking a glass of wine or beer regularly as part of a healthy lifestyle may be helpful for postmenopausal women.

"Everyone loses bone as they age, but not everyone develops osteoporosis," Turner said. "Being able to identify factors, such as moderate alcohol intake, that influence bone health will help people make informed lifestyle choices."

The study was funded by grants from the National Institutes of Health and the John C. Erkkila, M.D. Endowment for Health and Human Performance.

Karin Hardin, Adam Branscum, Kenneth Philbrick and Lynn Cialdella-Kam of OSU co-authored the study, along with Anne Breggia and Clifford Rosen of the Maine Medical Center Research Institute.

http://www.eurekalert.org/pub_releases/2012-07/uow-usd071112.php

UW scientists discover why human body cannot fight HIV infection

Gale Lab publishes results that could lead to new drug therapies

University of Washington researchers have made a discovery that sheds light on why the human body is unable to adequately fight off HIV infection. The work, directed by Dr. Michael Gale, Jr., a professor in the Immunology Department, will be featured in the August print issue of the Journal of Virology. The researchers discovered that the viral protein vpu, which is created by HIV during infection, directly interferes with the immune response protein IRF3 to dampen the ability of the immune system to protect against virus infection.

"By understanding exactly what HIV does to hamper the innate immune response during early infection, we can develop a clearer picture of how the virus is able to evade immunity to establish a long-term infection," said Dr. Brian Doehle, a postdoctoral fellow and lead author of the article. The research expanded on an earlier discovery by the Gale lab that HIV directly antagonizes the early innate immune response in infected cells by impairing IRF3 function.

The new studies found that the HIV protein vpu specifically binds to the immune protein IRF3 and targets it for destruction, thereby, preventing IRF3 from functioning to trigger an immune response within the infected cell.

The scientists also found that HIV strains engineered to lack vpu, which is made during infection, did not impair the immune response. "We have effectively identified a new Achilles heel in the arsenal that HIV uses to overcome the defenses present in the body's immune system", stated Dr. Gale. "This knowledge can be used to design new HIV antiviral therapeutics that prevent vpu from interacting with IRF3 and targeting it for destruction, thus enhancing immunity.

The development of new HIV antiviral therapeutics is critical to successfully treating HIV-infected people. Even though HIV antiviral therapeutics have already been developed and can effectively treat HIV infections, over time they lose their effectiveness due to the ability of the virus to adapt and spread despite the therapy, said Gale. "Therefore, the identification of new targets for treatment therapy is essential to providing the most effective treatment for HIV-infected patients". Gale's laboratory has already begun translating the knowledge from these discoveries to tracking the molecular events that occur in patients during infection.

Arjun Rustagi, an MD/PhD student in the UW Medical Scientist Training Program, has developed a procedure to measure IRF3 activity in human blood cells. This new methodology will be used to measure IRF3 function over the course of HIV infection -- from the early stages of acute infection to the later stages of chronic infection that lead to AIDS. By linking IRF3 function with infection over time, researchers will be able to understand how antiviral therapeutics that are designed to improve IRF3 function might impact the overall course of the disease in an HIV-infected individual.

Details on the development of the new assay will be published in the August 2012 issue of the journal, Methods.

The work was funded by grants from the National Institutes of Health. The laboratory of M. Juliana McElrath in the Vaccine and Infectious Disease Division at the Fred Hutchinson Cancer Research Center collaborated on the project. Results were published ahead of print on May 16 and May 30.

http://phys.org/news/2012-07-geoscientists-trigger-rapid-sea.html

Geoscientists discover trigger for past rapid sea level rise

The cause of rapid sea level rise in the past has been found by scientists at the University of Bristol using climate and ice sheet models.

The process, named 'saddle-collapse', was found to be the cause of two rapid sea level rise events: the Meltwater pulse 1a (MWP1a) around 14,600 years ago and the '8,200 year' event. The research is published today in Nature.Using a climate model, Dr Lauren Gregoire of Bristol's School of Geographical Sciences and colleagues unearthed the series of events that led to saddle-collapse in which domes of ice over North America became separated, leading to rapid melting and the opening of an ice free corridor. Evidence of these events has been recorded in ocean cores and fossil coral reefs; however, to date the reason behind the events was unclear and widely debated.

Ice domes up to 3 km thick (three times the height of Snowdon), formed in regions of high snowfall and higher topography, such as the Rocky Mountains. Together with the saddles – lower valleys of ice between the domes – these made up the ice sheet. Towards the end of the last ice age, at the time of mammoths and primitive humans, the climate naturally warmed. This started to melt ice at increasingly high elevations, eventually reaching and melting the saddle area between the ice domes. This triggered a vicious circle in which the melting saddle would lower, reach warmer altitudes and melt even more rapidly until the saddle had completely melted. In just 500 years, the saddles disappeared and only the ice domes remained. The melted ice flowed into the oceans leading to rapid sea level rises of 9 m in 500 years during the Meltwater pulse 1a event 14,600 years ago and 2.5 m in the second event, 8,200 years ago.

Dr Gregoire, lead author of the study, said: "We didn't expect our model to produce such a rapid sea level rise. We got really excited when we realised that the events we simulated corresponded to real events!"

In the model, Dr Gregoire found that saddle-collapse could explain a significant amount of the sea level rise observed: "The meltwater pulse produced by the saddle-collapse can explain more than half of the sea level jump observed around 14,600 years ago. The rest probably came from the progressive melting of ice sheets in Europe and Antarctica."

This research not only identifies the process which caused the melting of the North American ice sheet and the trigger for rapid sea level rises in the past, but also increases our understanding of the nature of ice sheets and climate change, allowing further questions to be posed and, with more research, answered. Research like this allows climate and ice sheet models to be tested against evidence from the real world. If climate models are able to reflect patterns observed in natural records our confidence in them increases. This is particularly relevant where the models are also used to investigate the effect of climate change on ice sheets in the future.

More information: Paper: 'Deglacial rapid sea level rises caused by ice sheet saddle collapses' by Lauren Gregoire, Antony Payne and Paul Valdes in Nature

http://www.sciencedaily.com/releases/2012/07/120711141855.htm

Switch Lets Early Lung Cancer Grow Unchecked

Cellular change thought to happen only in late-stage cancers to help tumors spread also occurs in early-stage lung cancer as a way to bypass growth controls

ScienceDaily - Cellular change thought to happen only in late-stage cancers to help tumors spread also occurs in early-stage lung cancer as a way to bypass growth controls, say researchers at Mayo Clinic in Florida. The finding, reported in the July 11 issue of Science Translational Medicine, represents a new understanding of the extent of transformation that lung cancer -- and likely many other tumor types -- undergo early in disease development, the scientists say. They add that the discovery also points to a potential strategy to halt this process, known as epithelial-mesenchymal transition, or EMT.

"Our study points to EMT as a key step in lung cancer progression during the earliest stages of cancer development," says lead investigator and cancer biologist Derek Radisky, Ph.D. "Normal cells recognize when they are dividing too rapidly, and turn on programs that block inappropriate cell division. Here we found that early-stage lung cancer cells switch on EMT in order to bypass these controls," he says.

The discovery could offer a new way to prevent progression to late-stage lung cancer, possibly by inhibiting a particular molecule from functioning, Dr. Radisky says. Because EMT is a well-recognized late-stage transition that occurs in all sorts of solid tumors, the researchers say they believe that the same early-stage use of EMT they found in lung cancer is likely occurring in other cancers.

EMT is a biological process used in embryonic development to allow body development, which requires the ability of cells and tissues to morph from one type to another, and develop in an orchestrated fashion.

Late-stage cancer uses EMT to change tumor cells into a form that can migrate through blood.

"The gaps in our knowledge of lung cancer have not allowed us to develop more effective targeted therapies," Dr. Radisky says. "This study offers us great new clues for a new approach to treating lung and possibly other cancers as early as possible."

Co-authors include researchers from Mayo Clinic in Rochester, Minn.; University Hospital Giessen and Marburg in Germany; Ontario Cancer Institute in Toronto; and the University of Colorado in Denver.

The study was funded by grants from the National Cancer Institute and the State of Florida's James & Esther King Biomedical Research Program.

Melody L. Stallings-Mann, Jens Waldmann, Ying Zhang, Erin Miller, Mona L. Gauthier, Daniel W. Visscher, Gregory P. Downey, Evette S. Radisky, Alan P. Fields, and Derek C. Radisky. Matrix Metalloproteinase Induction of Rac1b, a Key Effector of Lung Cancer Progression. Sci Transl Med, 11 July 2012 DOI: 10.1126/scitranslmed.3004062

http://www.wired.com/wiredscience/2012/07/scorpion-venom-antibiotic/

Scorpion Venom Heals Drug-Resistant Bacteria Infection

It may sound like snake oil, but a new study suggests scorpion venom contains a substance that can fend off drug-resistant bacteria, including the deadly MRSA.

By Tanya Lewis

Drug resistance is increasingly rendering our antibiotic arsenal ineffective against bacteria. According to a CDC study, MRSA caused 36 percent of staphylococcal infections in U.S. hospital intensive-care units in 1992, and as much as 64 percent of infections in 2003. But new research in mice suggests a solution may be hiding right under our feet.

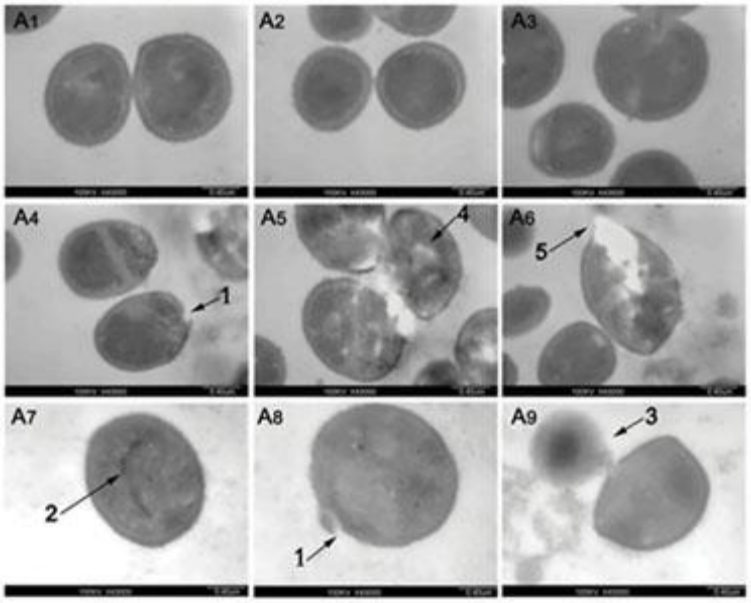
Many peptides (short strings of amino acids) found in many plants and animals have the ability to kill bacteria, fungi, viruses, and parasites. Virologists from China's Wuhan University took a peptide from the venom of a scorpion and modified it to strengthen its antibacterial activity. The modified peptide killed off both S. aureus and E. coli bacteria, and healed skin infections in mice, the study reported July 5 in the journal PLoS ONE.

The researchers gave mice skin infections and then treated some of them with the scorpion venom peptide. Those infections healed, while untreated infections or those treated with a placebo continued to fester. Under a microscope, skin treated with the venom peptide appeared normal again after four days, whereas the untreated mice suffered deep skin damage.

"They showed it's possible to take this peptide and turn it into an an antimicrobial peptide that can kill a broad range of bacteria that are harmful to humans," said immunologist Michael Zasloff of Georgetown University, who was not involved in the study. "The infections that we create on the backs of mice are not all that different from those that occur on humans."

Studying how bacteria grown outside the body responded to the peptide revealed its mechanism of attack. The peptide bound to substances in the bacteria's cell walls and coated them in tiny spheres, causing the bacteria to burst open and spill their contents (below).

Despite their efficacy, a common problem with antimicrobial peptides is their tendency to split open the infected animal's own blood cells in addition to bacterial cells.
The scorpion venom peptide is quite toxic, microbiologist Robert Hancock of the University of British Columbia, Canada, wrote in an email. But when the researchers tested the modified peptide with human red blood cells, it showed less of a tendency to split human cells than the original peptide had.
If the antibacterial effect works in humans, the new peptide could be a viable treatment for drug-resistant skin infections. Before that happens, it must be shown that the peptide can be produced cost-effectively, is safe to apply to human skin, and is effective in a clinical trial, said Zasloff.



Transmission electron microscopy of S. aureus bacteria with no treatment (top row), treated with the modified scorpion venom peptide (middle row), and treated with the original peptide (bottom row). **Cao et al (2012), PLoS ONE**

This peptide's clinical implications are merely a side effect of its original purpose: to prevent the scorpion's dinner from spoiling. "As far as a microbe is concerned, once a scorpion's prey is dead, it's like a piece of meat," said Zasloff. "It's what you would want to do if you didn't have a refrigerator."

http://arstechnica.com/science/2012/07/just-6-years-later-hpv-vaccine-may-already-provide-herd-immunity/

Just 6 years later, HPV vaccine may already provide herd immunity

Infection rates of the viruses it targets are dropping among the unvaccinated.

by John Timmer - July 12 2012, 6:30am TST

Many well-established vaccines are needlessly controversial, but a recently developed vaccine that targets the human papilloma virus (HPV) has some properties that caused unusual amounts of public discomfort. The vaccine itself doesn't actually prevent disease symptoms, since HPV infection goes largely unnoticed. Instead, it helps prevent a long-term outcome of infection: cervical cancer.
As the cancer's location might suggest, HPV is primarily spread through sexual contact, which is where the controversy comes in. For it to have the highest effectiveness, the vaccine must be administered before girls become sexually active, and thus at risk for HPV infection. To ensure this happens, public health authorities have recommended that girls receive the vaccine when they are 10-11 years old, an age that raises awkward questions about impending sexual activity with many parents. Meanwhile, boys are a common vector for the virus, but most recommendations have not suggested that they also be vaccinated.
Despite the public backlash (the vaccine actually became an issue in this year's Republican presidential campaign), many young women have been vaccinated, and a new study suggests that a form of herd immunity is already developing, in which the use of the vaccine is protecting women who haven't received it.
The study screened for the presence of HPV in two populations of sexually active teens and young women at two points: before the introduction of the vaccine in 2006, and four years after its introduction (in 2010). The populations were small (only about 400 at each time point), but still large enough to produce statistically significant results. A bigger problem was that the demographics of the two populations were quite different in terms of several factors associated with risk for HPV infection. The authors used an adjustment factor to control for this, but also provided the raw, unadjusted data.
Given that several clinical trials had shown that the vaccine is effective, it's no real surprise that the rate of infection with the four strains targeted by the vaccine (the ones most commonly associated with cervical cancer) went down significantly, dropping by about a third. (Many of the residual infections may result from not taking a complete course of the vaccine or having started it after the onset of sexual activity.) The big surprise is that the rate also dropped in those who hadn't been vaccinated, going from over 30 percent to under 20 percent. Adjusting for risk factors made the drop even more significant.
The authors suggest that this may be an indication of a very quick onset of a phenomenon called herd immunity. When there are fewer people around that carry an infectious agent, the chances of picking it up drop, so that even those who are not immune benefit from vaccination campaigns.

The vaccine itself only targets four strains of HPV, the ones most commonly associated with cancer. However, the researchers found that rates of infection with other strains had increased, going from roughly sixty percent to over 75 percent. The authors suggest this may be evidence that a concern voiced by some researchers may be playing out: the different strains of HPV compete with each other to some extent and, with the vaccine eliminating some of them, the remainder are spreading more efficiently. Fortunately, these other strains are less prone to causing cancer.

The population is small, and these results should be considered preliminary. But the possible indication of herd immunity is very promising news, even if the increased infection with other strains bears further monitoring. In the meantime, the authors reiterate that the numbers could be even better if more girls received a full vaccination before the age of 13. On the plus side, it's now recommended that boys be vaccinated as well, which should only enhance the herd immunity effect.

Three of the study's seven authors received funding for separate clinical trials from Merck, which developed the vaccine.
Pediatrics, 2012. DOI: 10.1542/peds.2011-3587 (About DOIs).

http://www.bbc.co.uk/news/health-18796309

Alzheimer's 'early signs timeline developed'

Scientists have assembled a "timeline" of the unseen progress of Alzheimer's before symptoms appear.

A team at Washington University School of Medicine looked at families with a genetic risk of the disease.

Writing in the New England Journal of Medicine, they say signs appeared up to 25 years before the expected onset of the disease.

UK experts said the ability to detect Alzheimer's early would give the best chance of successful treatment.

'Key changes'

The 128 people in the study, from the UK, US and Australia, had a 50% chance of inheriting one of three mutations that are certain to cause early Alzheimer's, which often develops in people's 30s and 40s - much earlier than the more common form of Alzheimer's which generally affects people in their 60s.

Those who carry the mutations will go on to develop the disease.

The researchers looked at the age the participants' parents were when they developed the disease - and therefore how many years it was likely to be before they too showed symptoms.

They underwent blood and spinal fluid tests as well as brain scans and mental ability assessments.

The earliest change - a drop in spinal fluid levels of the key ingredient of Alzheimer's brain plaques - can be detected 25 years before the anticipated age of disease onset, they suggest.

At 15 years, raised levels of tau, a structural protein in brain cells can be seen in spinal fluid - and shrinkage can also be detected within parts of the brain.

Changes in the brain's use of the sugar glucose and slight memory problems become apparent 10 years before symptoms would appear, they suggest.

Researchers also tested other members of the families without the inherited mutations - and found no changes in the markers they tested for.

Prof Clive Ballard, director of research at the Alzheimer's Society, said: "This important research highlights that key changes in the brain, linked to the inherited form of Alzheimer's disease, happen decades before symptoms show, which may have major implications for diagnosis and treatment in the future.

"These findings are a good indicator that there may be key changes in the brain happening early in people who develop non-hereditary Alzheimer's disease, but we can't be sure. Further research into this complex condition is needed to confirm a definite link."

And Dr Eric Karran, director of Research at Alzheimer's Research UK, said: "These results from people with the inherited form of Alzheimer's seem to be very similar to the changes in the non-genetic, common form of the disease.

"It's likely that any new treatment for Alzheimer's would need to be given early to have the best chance of success.

"The ability to detect the very earliest stages of Alzheimer's would not only allow people to plan and access care and existing treatments far sooner, but would also enable new drugs to be trialled in the right people, at the right time."

http://www.wired.com/wiredscience/2012/07/resistance-chicken-utis/

Investigation: Drug Resistance, Chicken And 8 Million UTIs

So, there's this thing. A big project. An investigative project, actually. I've been working on it for months, and finally I can tell you about it, because it all just published, in various venues, today.

By Maryn McKenna

I've been working with a great new group, the Food and Environment Reporting Network - one of the grant-funded journalism organizations that have arisen in the wake of the collapse of mainstream journalism - on an important, under-reported topic. Which is: Over the past decade, a group of researchers in several countries have been uncovering links between the use of antibiotics in chicken production and the rising occurrence of resistance in one of the most common bacterial infections in the world.

The infection in question is UTI, which just about every woman I know will recognize: It stands for urinary tract infection, and on average one out of every 9 women in the United States suffers one at least once per year. There are 6 million to 8 million UTIs in the US each year, costing at least $1 billion in healthcare spending. That's bad enough - not just the collective dollar amount, but the individual cost of annoyance, pain, lost work time to go to the doctor, and lost quality of life because of the urge, burning and disinclination to do anything except swill cranberry juice and spend hours in the bathroom. But an increasing percentage of UTIs are turning out to be antibiotic-resistant. That's potentially an even more serious problem, because when a UTI is not treated - or treated with drugs that do not work, which is effectively the same thing - the infection can climb up the urinary system to the kidneys, and from there cross to the bloodstream, and from there create a whole-body infection that can potentially be fatal. The organism that causes UTIs, which overwhelmingly is a particular form of E. coli known as ExPEC, already is believed to cause up to 40,000 deaths from bloodstream infections each year, and antibiotic resistance is expected to make that worse - or may be doing so already.

Antibiotic resistance in ExPEC E. coli has been climbing for years, and most people who treat such infections have had very little idea why. But this small group of researchers has persistently been proving connections between the resistance pattern in human UTIs, and the same resistance pattern in E. coli carried in chickens and turkeys before slaughter, and chicken and turkey meat sold in supermarkets in the U.S., Canada and Europe. They contend that the link among all of those is antibiotics given to chickens as they are raised, creating antibiotic-resistant organisms that move off the farm to imperil human health. And the more research they do, the stronger that link becomes.

This is such an important topic– so costly in dollars and quality of life, and so significant for what it says about the impact of antibiotic use in large-scale agriculture - that our investigation at FERN attracted several media partners.

We have ***a video package*** with ABC News on Good Morning America and ABC Nightly News (link coming for the evening news as the West Coast hasn't seen it yet) and a print partnership with The Atlantic that involves ***a much longer story*** than TV production limits allow.

From our story:

...the origin of these newly resistant E. coli has been a mystery - except to a small group of researchers in several countries. They contend there is persuasive evidence that the bacteria are coming from poultry. More precisely, coming from poultry raised with the routine use of antibiotics, which takes in most of the 8.6 billion chickens raised for meat in the U.S. each year.

Their research in the United States, Canada, and Europe (published most recently this month, in June, and in March) has found close genetic matches between resistant E. coli collected from human patients and resistant strains found on chicken or turkey sold in supermarkets or collected from birds being slaughtered. The researchers contend that poultry - especially chicken, the low-cost, low-fat protein that Americans eat more than any other meat - is the bridge that allows resistant bacteria to move to humans, taking up residence in the body and sparking infections when conditions are right.

"The E. coli that is circulating at the same time, and in the same area - from food animal sources, retail meat, and the E. coli that's causing women's infections - is very closely related genetically," said Amee Manges, Ph.D., an associate professor of epidemiology at McGill University in Montreal who has been researching resistant UTIs for a decade. "And the E. coli that you recover from poultry meat tends to have the highest levels of resistance. Of all retail meats, it's the most problematic that way."

Not everyone agrees with this interpretation of the science, of course, and if you go through to the Atlantic story, you'll find comments in it from several researchers who represent the agricultural research side and who don't agree.

When I do stories like this, involving deep dives into the medical literature, people always ask me what my sources are. Here (in addition to many interviews) are some of them:

Foodborne Pathogens and Disease, July 2012
Clinical Infectious Diseases, June 2012
Emerging Infectious Diseases, March 2012
Foodborne Pathogens and Disease, January 2012
Foodborne Pathogens and Disease, December 2011
The Lancet, July 2011
Emerging Infectious Diseases, June 2011
Clinical Microbiology and Infection, June 2011
Clinical Infectious Diseases, July 2009
Emerging Infectious Diseases, June 2007
Foodborne Pathogens and Disease, Spring 2005
Clinical Infectious Diseases, January 2005

There are others, but that seems like a good beginning for a bibliography.
This is an important, not well understood issue, and I hope you'll take a look.

Here's the Good Morning America segment, featuring George Stephanopoulos:

And here's ***the ABC World News segment***, featuring Diane Sawyer and ABC medical director Dr. Richard Besser, former acting director of the Centers for Disease Control and Prevention - and before that, chief of the CDC's efforts to track and reduce antibiotic resistance.
Updates:
The National Chicken Council disagrees with the investigation's results.
ABC News story on the investigation.
Twitter follower @JBetz03 alerts me to this additional newly published paper, from this month's Emerging Infectious Diseases, on strains of a different gut bacterium, E. faecalis, crossing between chickens and people in Vietnam.
NPR's Q&A with me about the backstory to the investigation.

http://www.eurekalert.org/pub_releases/2012-07/uadb-fed071212.php

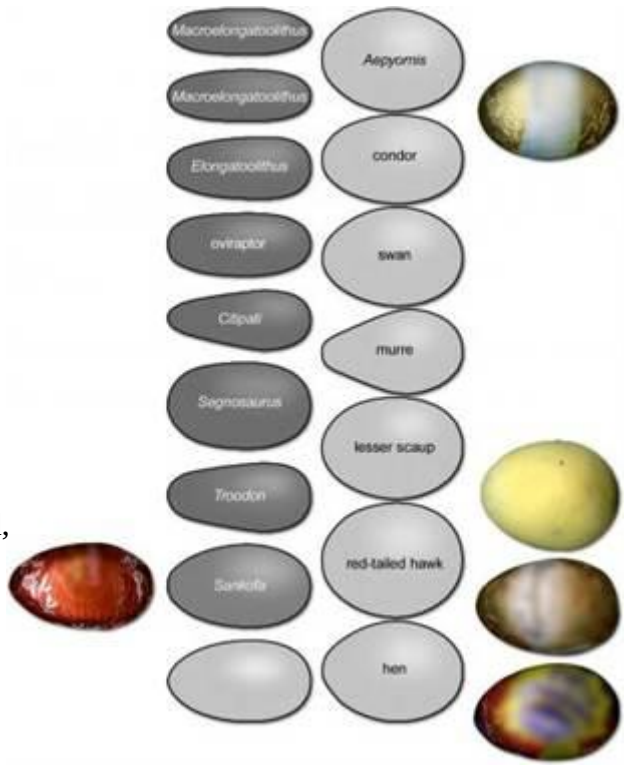
Fossil egg discovered in Lleida (Spain) links dinosaurs to modern birds

It is the only dinosaur egg in the world to have an oval shape, similar to that of chicken eggs

Before her death in December 2010, Nieves López Martínez, palaeontologist of the Complutense University of Madrid, was working on the research of dinosaur eggs with a very peculiar characteristic: an ovoid, asymmetrical shape. Together with Enric Vicens, palaeontologist of the Universitat Autònoma de Barcelona, the two scientists conducted an exhaustive analysis of their discovery, recently published in the journal Palaeontology.

The new type of dinosaur egg has been given the scientific name of Sankofa pyrenaica. The eggs were discovered in the Montsec area of Lleida, in two sites located on either side of the Terradets pass.

The South Pyrenean area is rich in dinosaur egg sites, most of which correspond to sauropod eggs from the upper Cretaceous, dating back more than 70 million years ago. During that period, the area was a coastal area full of beaches and deltas which won land from the sea through sediment accumulation. Sand and mud from that period gave way, millions of years later, to the sandstone and marl where dinosaur remains now can be found. On the beach ridges and flat coastal lands is where a large group of dinosaurs laid their eggs.



The pale gray eggs are from birds, and the darker gray eggs are from dinosaurs. Most Easter eggs, as shown on the right, are similar in shape to bird's eggs, but some are closer to the eggs of dinosaurs. The Easter egg on the left is particularly close to the newly described Sankofa dinosaur egg. **Mark Purnell / Univ. of Leicester**

The sites where the discoveries were made correspond to the upper Cretaceous, between the Campanian and Maastrichtian periods, some 70 to 83 million years ago. The fossils found belong to small eggs measuring some

7 centimetres tall and 4 cm wide, while the eggshell was on average 0.27mm thick. Most of the eggs found were broken in small fragments, but scientists also discovered more or less complete eggs, which can be easily studied in sections. The eggs found at the sites all belong to the same species. The main difference when compared to other eggs from the same period is their asymmetrical shape, similar to that of chicken eggs. The more complete samples clearly show an oval form rarely seen in eggs from the upper Cretaceous period and similar to modern day eggs.
Their shape is a unique characteristic of theropod eggs from the upper Cretaceous period and suggests a connection with bird eggs. Non avian dinosaur eggs are symmetrical and elongated. Asymmetry in bird eggs is associated to the physiology of birds: they take on this shape given the existence of only one oviduct which can form only one egg at a time. In this case the isthmus, the region in the oviduct creating the eggshell membrane, is what gives the egg its asymmetrical shape. Thanks to this shape, the wider end contains a bag of air which allows the bird to breathe in the last stages of its development. This evolutionary step was still relatively underdeveloped in dinosaurs.
Thus, the egg discovered by UCM and UAB researchers in certain manners represents the missing link between dinosaurs and birds. Only one other egg, discovered in Argentina and corresponding to a primitive bird from the same period, has similar characteristics. The discover represents proof in favour of the hypothesis that non avian theropods, the dinosaurs of the Cretaceous period, and birds could have had a common ancestor.

http://www.eurekalert.org/pub_releases/2012-07/ci-ssi070912.php

Solar system ice: Source of Earth's water

Scientists have long believed that comets and, or a type of very primitive meteorite called carbonaceous chondrites were the sources of early Earth's volatile elements

Washington, DC - Scientists have long believed that comets and, or a type of very primitive meteorite called carbonaceous chondrites were the sources of early Earth's volatile elements - which include hydrogen, nitrogen, and carbon—and possibly organic material, too. Understanding where these volatiles came from is crucial for determining the origins of both water and life on the planet. New research led by Carnegie's Conel Alexander focuses on frozen water that was distributed throughout much of the early Solar System, but probably not in the materials that aggregated to initially form Earth.
The evidence for this ice is now preserved in objects like comets and water-bearing carbonaceous chondrites. The team's findings contradict prevailing theories about the relationship between these two types of bodies and suggest that meteorites, and their parent asteroids, are the most-likely sources of the Earth's water. Their work is published July 12 by Science Express.
Looking at the ratio of hydrogen to its heavy isotope deuterium in frozen water (H2O), scientists can get an idea of the relative distance from the Sun at which objects containing the water were formed. Objects that formed farther out should generally have higher deuterium content in their ice than objects that formed closer to the Sun, and objects that formed in the same regions should have similar hydrogen isotopic compositions.
Therefore, by comparing the deuterium content of water in carbonaceous chondrites to the deuterium content of comets, it is possible to tell if they formed in similar reaches of the Solar System.
It has been suggested that both comets and carbonaceous chondrites formed beyond the orbit of Jupiter, perhaps even at the edges of our Solar System, and then moved inward, eventually bringing their bounty of volatiles and organic material to Earth. If this were true, then the ice found in comets and the remnants of ice preserved in carbonaceous chondrites in the form of hydrated silicates, such as clays, would have similar isotopic compositions.
Alexander's team included Carnegie's Larry Nitler, Marilyn Fogel, and Roxane Bowden, as well as Kieren Howard from the Natural History Museum in London and Kingsborough Community College of the City University of New York and Christopher Herd of the University of Alberta. They analyzed samples from 85 carbonaceous chondrites, and were able to show that carbonaceous chondrites likely did not form in the same regions of the Solar System as comets because they have much lower deuterium content. If so, this result directly contradicts the two most-prominent models for how the Solar System developed its current architecture. The team suggests that carbonaceous chondrites formed instead in the asteroid belt that exists between the orbits of Mars and Jupiter. What's more, they propose that most of the volatile elements on Earth arrived from a variety of chondrites, not from comets.
"Our results provide important new constraints for the origin of volatiles in the inner Solar System, including the Earth," Alexander said. "And they have important implications for the current models of the formation and orbital evolution of the planets and smaller objects in our Solar System."

This work was partially funded by NASA Cosmochemistry, the NASA Astrobiology Institute, Carnegie Institution of Canada, the Natural Sciences and Engineering Research Council of Canada , the W.M. Keck Foundation , and the UK Cosmochemical Analysis Network. For supplying the many samples that were necessary for this work, the authors would like to thank: the members of the Meteorite Working Group; Cecilia Satterwhite and Kevin Righter (NASA, Johnson Space Center); Tim McCoy and Linda Welzenbach (Smithsonian Museum for Natural History); Laurence Garvie (Arizona State University); Sara Russell, Caroline Smith, and Deborah Cassey (Natural History Museum, London).

http://www.eurekalert.org/pub_releases/2012-07/cp-mso070812.php

Male sex ornaments are fishing lures, literally

Talk about a bait-and-switch.

Male representatives of the tropical fish known as swordtail characins have flag-like sex ornaments that catch mates just like the bait on a fishing rod would. What's more, a study reported online on July 12 in Current Biology, a Cell Press publication, shows just what any good fly-fisherman would know: Lures work best if they mimic the foods that fish most often eat. For some characins in the study, that means males are waving pretend ants around in hopes of getting a bite.

"This is a natural example of a fishing lure designed to maximize the chance to catch a fish," said Niclas Kolm of Uppsala University. "In this case, it is not just any fish, however—it is a fish of the opposite sex that the lure is designed to catch."

The findings also lend support to the notion that sensory drive can encourage fish and other animals to diversify and ultimately to become separate species. Sensory drive is the idea that communication signals will work best when they are a good match for their surroundings and that they will diversify when those surroundings vary.

The characins living in Trinidad show considerable variation in the shape of male sex ornaments, and the researchers suspected that those differences might have something to do with what they eat. The fish mainly eat bugs, including ants, beetles, springtails, and fly larvae, which fall onto the water surface. In some populations, characins eat mostly ants, while in others they eat few.

Kolm's team now confirms that characins that mainly eat ants also carry sex ornaments that look more like ants. Studies in the lab also show that females who have been fed on ants prefer to bite at the ornaments of males from populations that mainly eat ants. That's presumably because those females rapidly develop a search image for ants.

The findings show that sensory drive can promote differences among populations based entirely on other species in the community, even when all other environmental factors are the same. They also blur the distinction between food and mate preferences, the researchers say.

In this case, it seems, the best mate is also the one that looks most like dinner.

Kolm et al.: "Diversification of a food-mimicking male ornament via sensory drive."

http://bit.ly/LlvHtG

Hominins did not need boats to settle islands

The early human colonisation of islands might not have been plain sailing.

12:00 12 July 2012 by Jeff Hecht

Instead of using boats to deliberately settle on Indonesian islands, hominins may have arrived as castaways, carried on floating debris after floods.

David Wilkinson of Liverpool John Moores University and Graeme Ruxton of the University of St Andrews, both in the UK, used population estimates from the early settlement of Polynesia to model the likely success of island settlement attempts in human prehistory.

They found that five young couples had a 40 per cent chance of giving rise to a population of 500 – or founding a population that survived for 500 years. Ten random castaways had only a 20 per cent chance of similar success. But throwing in between one and four additional castaways every 50 years raised the chances of an accidental settlement succeeding to 47 per cent.

Stone tools show that hominins – possibly Homo erectus – reached Flores 1 million years ago. The famous Homo floresiensis may have descended from this population of H. erectus. Meanwhile, a study earlier in the year concluded that Neanderthals had access to boats 100,000 years ago, which they used to reach the Greek islands of Lefkada, Kefallonia and Zakynthos, where their stone tools have been found.

The new finding suggests that in both cases the hominins could have reached the islands without boats. We already know that other mammals managed the feat: rats and Stegodon, an extinct relative of the elephant, crossed the deep-water channel between the Indonesian islands of Java and Flores. Elephants are strong swimmers, and rats could have travelled on storm debris.

Accidental colonisation of Flores by hominins would have been difficult, but not impossible, says Mike Morwood of the University of Wollongong in New South Wales, Australia. However, he adds, "the rapid colonisation of Greater Australia and major islands of western Melanesia 45,000 to 50,000 years ago indicates deliberate colonisation voyages by people using directed craft".

Journal of Human Evolution, DOI: 10.1016/j.jhevol.2012.05.013

http://phys.org/news/2012-07-graphene-holes-automatically.html

New research shows that graphene is able to seal holes in itself automatically

New research shows that graphene is able to seal holes in itself automatically

July 12, 2012 by Bob Yirka report

Phys.org - Graphene, the one carbon atom thick sheet material has been in the news so much over the past couple of years it would seem that a saturation point has been reached. But then along comes another new discovery that is not only newsworthy, but offers hope that one day soon scientists will be able to figure out a way to mass produce the stuff allowing the full potential of the "miracle" material to finally emerge. The new discovery has been made by a team at the University of Manchester, one of whose members include Konstantin Novoselov, co-winner of the Nobel Prize for his work in discovering graphene. The team has found that when holes are made in a graphene sheet, if carbon atoms are brought near, they will be snapped up by the graphene sheet, repairing the hole and returning the sheet to its original properties. A paper written by the team was first uploaded to the preprint server arXiv, then shortly thereafter was published in Nano Letters.

Researchers have been excited about graphene because it offers so much promise in such a wide variety of applications ranging from electronics to advanced computers to medical applications. What's been holding back the development of applications that can take advantage of its special properties though, has been the difficulty in mass producing it; being just one atom thick it tends to break easily and curl up into a ball. Because of this, a lot of research has been going on trying to figure out a way to get around both problems, and that's exactly what the research team at Manchester was doing when they discovered a new surprising property; graphene can heal itself under the right conditions.

The team used a combination of an electron beam and metals such as palladium or nickel to create tiny stable holes (to attach electrodes) in a sheet of graphene that had been previously grown. In so doing they found that adding more metal allowed them to make larger holes that would remain stable, a significant find itself. But then, when they added carbon to the mix, they found that the carbon atoms displaced the metal and in the process caused the hole to be sealed, effectively returning the sheet to its original pristine condition. The researchers also found that if they used a hydrocarbon, the hole was sealed as well, but left a bumpy scar.

The teams' results offer more than just a surprise, because at this juncture it seems possible that by manipulating this newly found property (such as using it as part of the growth process), a way might be found to mass produce the material, finally unleashing its full potential.

More information: Graphene Reknits Its Holes, Nano Lett., Article ASAP. DOI: 10.1021/nl300985q

Abstract

Nanoholes, etched under an electron beam at room temperature in single-layer graphene sheets as a result of their interaction with metal impurities, are shown to heal spontaneously by filling up with either nonhexagon, graphene-like, or perfect hexagon 2D structures. Scanning transmission electron microscopy was employed to capture the healing process and study atom-by-atom the regrown structure. A combination of these nanoscale etching and reknitting processes could lead to new graphene tailoring approaches.

Available on: arXiv:1207.1487v1 [cond-mat.mes-hall] arxiv.org/abs/1207.1487 Journal reference: arXiv Nano Letters

http://bit.ly/Nt38fC

FBI forensic review could free thousands of prisoners

Thousands of people jailed on the basis of forensic evidence could walk free as the US criminal justice system looks to bring forensics in line with modern science.

18:15 12 July 2012 by Sara Reardon

The Federal Bureau of Investigation and the US Department of Justice announced this week that they will review more than 10,000 criminal cases dating back to 1985. These are instances in which guilty verdicts were based on forensic hair and fibre analyses, along with other methods that no longer stand up to scientific scrutiny. The review follows numerous investigations that have cast doubt on the validity of most forensic methods. Firearms and bullet analysis, for instance, can wrongly place innocent people at a crime scene.

In a strongly worded 2009 report, the US National Academy of Sciences (NAS) said that with the exception of nuclear DNA evidence, "no forensic method has been rigorously shown to have the capacity to consistently, and with a high degree of certainty, demonstrate a connection between evidence and a specific individual". But even DNA evidence is fallible: different labs can return very different results for the same sample.

Paul Cates of the Innocence Project in New York, which campaigns for people wrongly convicted on the basis of DNA evidence, says that to date, 292 people have had their conviction overturned by follow-up DNA analyses. "In half of those cases, there had been some sort of forensics problem," he says.
Cates backs a recommendation in the NAS report, which called for a national body to set standards for forensic science so that all analysts would be consistent. "We're hopeful that Congress will take up this cause in short order to ensure that what passes as forensics is science," says Cates.

http://www.sciencedaily.com/releases/2012/07/120712144749.htm

Discovery of Chemical That Affects Biological Clock Offers New Way to Treat Diabetes

Biologists discovered a chemical that offers a completely new direction for the development of drugs to treat metabolic disorders such as type 2 diabetes

ScienceDaily - Biologists at UC San Diego have discovered a chemical that offers a completely new and promising direction for the development of drugs to treat metabolic disorders such as type 2 diabetes -- a major public health concern in the United States due to the current obesity epidemic.
Their discovery, detailed in a paper published July 13 in an advance online issue of the journal Science, initially came as a surprise because the chemical they isolated does not directly control glucose production in the liver, but instead affects the activity of a key protein that regulates the internal mechanisms of our daily night and day activities, which scientists call our circadian rhythm or biological clock.
Scientists had long suspected that diabetes and obesity could be linked to problems in the biological clock. Laboratory mice with altered biological clocks, for example, often become obese and develop diabetes. Two years ago, a team headed by Steve Kay, dean of the Division of Biological Sciences at UC San Diego, discovered the first biochemical link between the biological clock and diabetes. It found that a key protein, cryptochrome, that regulates the biological clocks of plants, insects and mammals also regulates glucose production in the liver and that altering the levels of this protein could improve the health of diabetic mice.
Now Kay and his team have discovered a small molecule -- one that can be easily developed into a drug -- that controls the intricate molecular cogs or timekeeping mechanisms of cryptochrome in such a manner that it can repress the production of glucose by the liver. Like mice and other animals, humans have evolved biochemical mechanisms to keep a steady supply of glucose flowing to the brain at night, when we're not eating or otherwise active.
"At the end of the night, our hormones signal that we're in a fasting state," said Kay. "And during the day, when we're active, our biological clock shuts down those fasting signals that tell our liver to make more glucose because that's when we're eating."
Diabetes is caused by an accumulation of glucose in the blood, which can lead to heart disease, strokes, kidney failure and blindness. In type 1 diabetes, destruction of insulin producing cells in the pancreas results in the high blood sugar. In type 2 diabetes, which makes up 90 percent of the cases, gradual resistance to insulin because of obesity or other problems, leads to high blood sugar.
Kay and his collaborators discovered in 2010 that cryptochrome plays a critical role in regulating the internal timing of our cyclical eating patterns, timing our fasting at night with our eating during the day to maintain a steady supply of glucose in our bloodstream. Other researchers have recently discovered that cryptochrome also has the potential to reduce high blood sugar from asthma medication by adjusting the time of day a patient takes their medication. "We found that if we increased cryptochrome levels genetically in the liver we could inhibit the production of glucose by the liver," said Kay.
What he and his team found in their most recent discovery was that a much smaller molecule, dubbed "KL001" (for the first such compound from the Kay Lab), can regulate that activity as well. It slowed down the biological clock by stabilizing the cryptochrome protein -- that is, it essentially prevented crypotochrome from being sent to the cellular garbage can, the proteasomes.
The discovery of KL001 was serendipitous, a complete surprise to the scientists that came about from a parallel effort in Kay's laboratory to identify molecules that lengthen the biological clock. Two years ago, Tsuyoshi Hirota, a postdoctoral fellow in Kay's laboratory found a compound that had the greatest effect ever seen on circadian rhythm, a chemical the biologists dubbed "longdaysin" because it lengthened the daily biological clocks of human cells by more than 10 hours.
Continuing his search, Hirota resumed his efforts to find more chemicals that lengthened or slowed down circadian rhythms, enabling the scientists to understand more about the intricate chemical and genetic machinery of the biological clock. He and his colleagues in Kay's lab did this by screening thousands of compounds from a chemical library with human cells in individual micro-titer wells in which a luciferase gene from fireflies is attached to the biological clock machinery, enabling the scientists to detect a glow whenever

the biological clock is activated. Their molecular fishing expedition came up with a number of other compounds, one of which was KL001.
"We found other compounds that like longdaysin slowed down the biological clock," said Kay. "But unlike longdaysin, these compounds did not inhibit the protein kinases that longdaysin inhibits so we knew this compound must be working differently. What we needed to know was what is this compound interacting with? And we were absolutely stunned when we discovered that what was binding most specifically to our compound, KL001, was the clock protein cryptochrome that our lab has worked on in plants, flies and mammals for the last 20 years."
Kay's team turned to biological chemists in Peter Schultz's laboratory at The Scripps Research Institute to characterize the compound and understand better chemically how it affected cryptochrome to lengthen the biological clock.
"Those biochemical studies showed us that KL001 prevents cryptochrome from being degraded by the proteasome system, which was another big surprise," said Kay. "It essentially interferes with the signal to send cryptochrome to the garbage can."
To understand how KL001 worked mechanistically with cryptochrome to control the biological clock, the team initiated a collaboration with Frank Doyle and his group at UC Santa Barbara. "They constructed a beautiful mathematical model of cryptochrome's role in the clock," said Kay. "That model was essential in allowing us to understand the action of the compound because the biological clock is very complicated. It's like opening the back of a Rolex and seeing the hundreds of tiny little cogs that are tightly integrated."
Based on that mathematical model, the scientists predicted that adding KL001 to mouse liver cells should stabilize cryptochrome and that the increased level of cryptochrome would inhibit the production of enzymes in the liver that stimulate the process of gluconeogenesis -- the generation of glucose during fasting. Experiments done together with the laboratory of David Brenner, dean of the UC San Diego School of Medicine and Vice Chancellor for Health Sciences, confirmed that prediction to be true.
"In mouse liver cells," said Kay, "we showed that KL001 inhibited gene expression for gluconeogenesis that is induced when exposed to the hormone glucagon, which promotes glucose production by the liver. It's a hormone we all produce in fasting states. And our compound, in a dose dependent way, inhibits hepatic gluconeogenesis, the actual production of glucose by those liver cells."
Kay said the next step for the research group is to understand how KL001 and similar molecules that affect cryptochrome function in living systems, such has laboratory mice. The scientists also plan to probe how such compounds affect other processes besides the liver that may tie the biological clock to metabolic diseases. "As with any surprise discovery," he notes, "this opens the door to more opportunities for novel therapeutics than we can currently imagine."
Besides Kay, Hirota, Schultz, Doyle and Brenner, other scientists involved in the discovery were Mariko Sawa, Pagkapol Y. Pongsawakul and Tim Sonntag of UC San Diego's Division of Biological Sciences; Jae Wook Lee of TSRI; Peter St. John of UC Santa Barbara; Keiko Iwaisako, Takako Noguchi and David Welsh of UC San Diego's School of Medicine.
The study was supported by grants from the National Institutes of Health (GM074868, H051573 GM085764, GM096873, MH082945).

http://nyti.ms/SAIY70

In Preventing Alzheimer's, Mutation May Aid Drug Quest

A study of a rare gene mutation that protects people against Alzheimer's disease provides the strongest evidence yet that excessive levels of beta amyloid are a driving force in the disease

By GINA KOLATA

A study of a rare gene mutation that protects people against Alzheimer's disease provides the strongest evidence yet that excessive levels of a normal brain substance, beta amyloid, are a driving force in the disease - bolstering hopes that anti-amyloid drugs already under development might alter the disease's course or even prevent it.
DeCode Genetics stored blood samples from its research on genomes and Alzheimer's disease.
So far, the drugs have not succeeded. But scientists not connected with the new study said it suggested that the drug companies' big bets on anti-amyloid treatments could yet pay off.
The implication for drug development "is hugely important," said Dr. David Altshuler, a genomics expert at Harvard Medical School and the Broad Institute of Harvard and M.I.T.
And Dr. Samuel Gandy, an Alzheimer's researcher who directs the Mount Sinai Center for Cognitive Health, called the finding the most significant in the field in two decades, since researchers first reported a mutation that leads to the disease.

The protective mutation, whose discovery was reported online Wednesday in the journal Nature, is highly uncommon — it is not the reason most people do not develop Alzheimer's. But what intrigues researchers is how it protects the brain.

Mutations that cause Alzheimer's lead to excessive amounts of beta amyloid in the brain; by contrast, the protective mutation slows beta amyloid production, so people make much less. "This paper provides strong evidence that it would work in the general population if you did it right," Dr. Altshuler said.

Scientists at the drug companies agreed. "We are thrilled," said Ryan Watts, one of the authors of the new paper and head of the neurodegeneration labs at Genentech, which is developing two drugs to reduce brain amyloid levels.

Dr. Richard Mohs, leader of neuroscience early clinical development at Eli Lilly, said the company was "very encouraged by these study results." They show, he said, that despite an initial failure, the strategy of focusing on drugs to reduce beta amyloid levels is "a logical path for the development of effective therapies that may slow disease progression."

Many questions remain, of course. Most people do not have the protective gene mutation, but as common as Alzheimer's is, most people do not get it. It is not clear why. And most who develop Alzheimer's do not have one of the rare gene mutations that cause it. The reasons for their disease are unclear.

The discovery of the protective gene mutation, a product of the revolution that has taken place in genetics, arose when researchers scanned the entire DNA of 1,795 Icelanders.

About 1 in 100 had a mutation in the gene for a large protein that is sliced to form beta amyloid. Then the investigators studied people who had been given an Alzheimer's diagnosis, and a group of people 85 and older. Those with the mutation appeared to be protected from Alzheimer's disease.

The investigators, led by Dr. Kari Stefansson, chief executive at DeCode Genetics, an Icelandic company, looked at genomes of North Americans and found the gene mutation in only about 1 in 10,000 people. That indicates, Dr. Stefansson said, that the mutation arose relatively recently in Scandinavia.

The protective gene even appears to override a very strong risk factor for Alzheimer's disease in old age — two copies of a gene known as ApoE4. Ninety percent of people with two ApoE4 genes get Alzheimer's by age 80. But Dr. Stefansson says there are 25 people in his study with two copies of ApoE4. None have Alzheimer's disease.

The research "is obviously right," said John Hardy, an Alzheimer's researcher at University College London and a discoverer of the first gene mutation found to cause the disease. "The statistics and the finding are pretty secure."

The discovery is part of a continuing story that implicates beta amyloid as a central and crucial player in this destructive brain disease. The idea began two decades ago with the discovery of very rare gene mutations that always cause Alzheimer's in those who inherit them, usually by middle age. The mutations were different in different families, but all had the same effect: They increased the amount of beta amyloid in the brain. That meant that a buildup of amyloid was sufficient to cause the disease.

Elderly people with Alzheimer's — who typically do not have these gene mutations — also had excess amyloid in the brain. So researchers reasoned that might mean that excess amyloid was causing the disease in them, too.

Additional evidence of the role of beta amyloid was reported on Wednesday in The New England Journal of Medicine. Using spinal taps and brain scans to track the protein, investigators found that people with one of the Alzheimer's-causing mutations start making too much beta amyloid as long as 20 years before they have symptoms of the disease.

Researchers and drug companies focused on the amyloid hypothesis to the extent that almost every experimental drug being tested to slow or halt Alzheimer's disease is designed to reduce the amount of amyloid in the brain. Most of those drugs are still being tested in clinical trials, but a Lilly drug that failed spectacularly in 2010, semagacestat, actually made people with Alzheimer's worse and gave rise to soul-searching in the field. It emphasized a crucial question that hung over the endeavor. Was amyloid really causing Alzheimer's in elderly people? Might the protein instead be a bystander, accumulating, for example, as part of the brain's response to damage?

The discovery of the protective gene mutation provides strong clues. People with the mutation make substantially less beta amyloid, but other than that they are no different from anyone else. And they do not get Alzheimer's.

People could be tested to see if they have the protective mutation, Dr. Stefansson said, but he added, "The benefits of doing so are not obvious to me." He explained that since the gene is so rare, chances that a person being tested would have it - especially if that person is not Scandinavian - are extremely low. Almost everyone

would end up with the same uncertainty they have now. There is as yet no way to prevent Alzheimer's and, outside of families with one of the rare disease-causing gene mutations, no way to know who is going to get it. Still, Dr. Hardy noted, as provocative as the discovery of the protective gene mutation is, the strategy of reducing amyloid levels - the ultimate test of the amyloid hypothesis - still must be evaluated in typical Alzheimer's disease. For example, perhaps people need to have lower levels of beta amyloid from birth to really be protected.

Researchers and companies explain away the failure of the first few experimental drugs to reduce beta amyloid levels or to block the protein by saying they were not powerful enough and were studied in people who already had the disease and clear symptoms of mental decline. By then it might be too late to make any difference. When brain cells have died, nothing can bring them back.

The strategy now is to use new brain scans and other methods to find and treat people before they have symptoms of mental decline.

"The idea is that treatment has to start early to make a difference," Dr. Watts said.

Of course, people with the newly discovered mutation have lower levels of beta amyloid for their entire lives. "You couldn't start earlier than that," Dr. Watts said.

http://www.scientificamerican.com/article.cfm?id=marijuana-reveals-memory-mechanism

Marijuana Reveals Memory Mechanism

Glial cells, not neurons, are responsible for marijuana-induced forgetfulness

By Ruth Williams | July 13, 2012 | 16

Until recently, most scientists believed that neurons were the all-important brain cells controlling mental functions and that the surrounding glial cells were little more than neuron supporters and "glue." Now research published in March in Cell reveals that astrocytes, a type of glia, have a principal role in working memory. And the scientists made the discovery by getting mice stoned.

Marijuana impairs working memory—the short-term memory we use to hold on to and process thoughts. Think of the classic stoner who, midsentence, forgets the point he was making. Although such stupor might give recreational users the giggles, people using the drug for medical reasons might prefer to maintain their cognitive capacity.

To study how marijuana impairs working memory, Giovanni Marsicano of the University of Bordeaux in France and his colleagues removed cannabinoid receptors—proteins that respond to marijuana's psychoactive ingredient THC—from neurons in mice. These mice, it turned out, were just as forgetful as regular mice when given THC: they were equally poor at memorizing the position of a hidden platform in a water pool. When the receptors were removed from astrocytes, however, the mice could find the platform just fine while on THC.

The results suggest that the role of glia in mental activity has been overlooked. Although research in recent years has revealed that glia are implicated in many unconscious processes and diseases [see "The Hidden Brain," by R. Douglas Fields; Scientific American Mind, May/June 2011], this is one of the first studies to suggest that glia play a key role in conscious thought. "It's very likely that astrocytes have many more functions than we thought," Marsicano says. "Certainly their role in cognition is now being revealed."

Unlike THC's effect on memory, its pain-relieving property appears to work through neurons. In theory, therefore, it might be possible to design THC-type drugs that target neurons—but not glia—and offer pain relief without the forgetfulness.

http://bit.ly/LsvxWa

Growth of Earth's core may hint at magnetic reversal

Lopsided growth of the Earth's core could explain why its magnetic field reverses direction every few thousand years.

15:29 13 July 2012 by Will Ferguson

If it happened now, we would be exposed to solar winds capable of knocking out global communications and power grids. One side of Earth's solid inner core grows slightly while the other half melts. Peter Olson and Renaud Deguen of Johns Hopkins University in Baltimore, Maryland, used numerical modelling to establish that the axis of Earth's magnetic field lies in the growing hemisphere – a finding that suggests shifts in the field are connected to growth of the inner core.

There are signs that the next switch may be under way: rapid movements of the field's axis to the east in the last few hundred years may be a precursor to the north and south poles trading places, the researchers speculate.

"What we found that is interesting in our models is a correlation between these transient [shifts] and reversals [of Earth's magnetic field]," says Olson. "We kind of speculate there is that connection but the chaos in the core is going to prevent us from making accurate predictions for a long time."

Bruce Buffett of the University of California, Berkeley, says the authors present an intriguing proof of concept with their model. "They are suggesting very cautiously that maybe this rapid change is somehow suggestive of us going into a reversal event," he says. "You could imagine if the field were to collapse it would have disastrous consequences for communication systems and power grids."

Journal reference: Nature Geoscience, DOI: 10.1038/ngeo1506

http://phys.org/news/2012-07-million-year-storage-solution-stone.html

Million-year storage solution is set in stone

A sapphire hard disk can last one million years and resolve a problem worrying archaeologists.

Phys.org - Thursday, Patrick Charton of the French nuclear waste management agency ANDRA, presented a way out of data storage problems, an information-engraved sapphire disk using platinum. The disk is being called the ultimate, if not ultimately unaffordable, HDD. The disk was announced at this week's Euroscience Open Forum, a pan-European event drawing researchers, as a way to provide information for future archaeologists.

The solution is in the form of two thin disks of industrial sapphire, molecularly fused, with a thin layer of inscribed platinum. The disks were immersed in acid to test their durability and to simulate aging.

With the sapphire disk, up to 40,000 miniaturized pages of text or images etched can be inscribed in the platinum. The information would be read with microscope.

A key application would be as a solution for how future societies will be able to identify areas of buried nuclear waste. Nuclear reactors produce radioactive waste that needs to be safely stored for up to one million years. Once a disposal method is determined, future societies will need to know where the waste is buried.

According to Science magazine. Finland, France, and Sweden are the furthest advanced in the process of finding a geologically suitable site. While designers of such repositories are confident the waste can be buried safely, the fear is that future archaeologists may dig in t he wrong places. Markers would be a way to allow them to know the sites where they should not dig.

With a sapphire disk, the warning message could be encoded into varied forms of written human communication, including words, pictograms, and diagrams, and in turn linguists and artists are involved in the project. The researchers say thus far they have no idea what language to use. Another application is seen is as a Rosetta Stone to preserve the wealth of knowledge that humans have accumulated. The prototype shown costs $30,000 to make.

Euroscience Open Forum is a meeting drawing scientists, researchers, policy makers, and the general public. Talks focus on the direction that research is taking in the sciences, humanities and social science.

http://www.bbc.co.uk/news/uk-scotland-tayside-central-18827752

Study says chimpanzees use 'human-like gestures'

Wild chimpanzees communicate using similar gestures to humans, according to a Stirling researcher.

Dr Anna Roberts said she had identified about 20 to 30 manual gestures used by chimps, up to a third of which were similar to those used by humans. The chimps' gestures included beckoning to make someone approach or flailing their arms to make someone leave. It is hoped the finding may help researchers understand how humans evolved language.

Dr Roberts studied chimpanzees in the wild in Uganda over an eight-month period. She believes the gestures suggest that a common ancestor of humans and chimpanzees must have used similar manual gestures.

'**Mind-reading**'

The Stirling University scientist said: "Chimpanzees use these gestures intentionally to elicit a desired response from other chimpanzees and they may be the missing link between ape and human communication. "We now know that these gestures must have been in the repertoire of our common ancestor and might have been the starting point for language evolution." The study found the animals used gestures to communicate a range of activities including nursing, feeding, sex, aggression and defence.

Dr Roberts also discovered that chimpanzees not only communicate using manual gestures, but are able to work out what the signaller means from both gesture and accompanying context.

She said: "The defining way that people understand communication with others is by figuring out what someone really means by 'mind-reading' their intentions and we have discovered that chimpanzees may have a similar ability."

Dr Roberts said the research showed that the basic elements for the evolution of language appeared to be present in our closest living relatives.

http://www.sciencedaily.com/releases/2012/07/120713140200.htm

Large-Animal Biocontainment Laboratory Needed to Protect Animal and Public Health, Experts Urge

It is "imperative" that the U.S. build a large-animal biocontainment laboratory to protect animal and public health, says a new report by the National Research Council.

ScienceDaily - Two options that could meet long-term needs include the National Bio- and Agro-Defense Facility (NBAF) facility as currently designed, or a scaled-back version tied to a distributed laboratory network. Until such a facility opens that is authorized to work with highly contagious foot-and-mouth disease, the Plum Island Animal Disease Center located off Long Island should remain in operation to address ongoing needs. The report concludes that there are important drawbacks for the U.S., should it rely solely on international laboratories to meet large animal Biosafety Level 4 needs in the long term.

The proposed NBAF in Manhattan, Kan., would be the world's fourth Biosafety Level 4 laboratory capable of large animal research and would replace the aging Plum Island facility. NBAF would study highly contagious foreign animal diseases -- including foot-and-mouth disease, which affects cattle, pigs, deer, and other cloven-hoofed animals -- as well as emerging and new diseases that can be transmitted between animals and people. However, given the estimated cost of $1.14 billion to construct NBAF at the proposed site and the country's current fiscal challenges, the U.S. Department of Homeland Security requested that the National Research Council analyze whether three options could meet the nation's laboratory infrastructure needs.

The three options as stipulated by DHS were: constructing NBAF as designed, constructing a "scaled-back" version of NBAF, and maintaining current capabilities at Plum Island Animal Disease Center. Because the Plum Island facilities do not have large animal Biosafety Level 4 capacity -- containment of agents that are potentially life-threatening to humans and pose a high risk of transmission -- this type of work would have to be conducted at foreign laboratories.

The scope of the committee's analysis was limited to examining the three options and explicitly excluded an assessment of specific site locations for the proposed laboratory facility; therefore, the report neither compares relative risks of the three options nor determines where foot-and-mouth disease research can be safely conducted. In addition, the committee concluded that to most appropriately fill laboratory needs, all factors of concern will need to be considered in a more comprehensive assessment.

The report concludes that DHS' first option -- NBAF as currently designed -- includes all components of the ideal laboratory infrastructure in a single location and has been designed to meet the current and anticipated future mission needs of DHS and the U.S. Department of Agriculture's Agricultural Research Service and Animal and Plant Health Inspection Service. However, the proposed facility also has drawbacks, including substantial costs associated with construction, operation, and management; and not leveraging existing capacity at other containment laboratories in the U.S.

Regarding the second option, the report finds that a partnership between a central national laboratory of reduced scope and size and a distributed laboratory network can effectively protect the United States from foreign animal and zoonotic diseases, potentially realize cost savings, reduce redundancies while increasing efficiencies, and enhance the cohesiveness of a national system of biocontainment laboratories. However, the cost implications of reducing the scope and capacity of a central facility are not known.

In its assessment of the third option, the report says that maintaining the Plum Island Animal Disease Center and leveraging foreign laboratories for large animal Biosafety Level 4 needs would avoid the costs of constructing a new replacement facility. However, the facilities at Plum Island do not meet current standards for high biocontainment. Given the uncertainty over priorities of a foreign laboratory and logistical difficulties in an emergency, it would not be desirable for the United States to rely on international laboratories to meet these needs in the long term. The report adds that because foot-and-mouth disease research remains critical for the U.S. animal health system, it will be essential to maintain the Plum Island facility until an alternative facility is authorized, constructed, commissioned, and approved for work with the virus.

Regardless of the options considered for a central facility, the report recommends that DHS and USDA develop and implement an integrated national strategy that utilizes a distributed system for addressing foreign animal and zoonotic disease threats. The capital costs associated with maintaining or constructing modern laboratory facilities should be balanced with the need to support research priorities. Therefore, it is critical for DHS and USDA to develop solutions that strike a balance between facilities costs and the research and development effort needed to protect American agriculture and public health.

Meeting Critical Laboratory Needs for Animal Agriculture: Examination of Three Option: http://www.nap.edu/catalog.php?record_id=13454

http://nyti.ms/P9Wz4S

The Ecology of Disease

If we fail to understand and take care of the natural world, it can cause a breakdown of these systems and come back to haunt us in ways we know little about.

By JIM ROBBINS

THERE'S a term biologists and economists use these days - ecosystem services - which refers to the many ways nature supports the human endeavor. Forests filter the water we drink, for example, and birds and bees pollinate crops, both of which have substantial economic as well as biological value.

If we fail to understand and take care of the natural world, it can cause a breakdown of these systems and come back to haunt us in ways we know little about. A critical example is a developing model of infectious disease that shows that most epidemics — AIDS, Ebola, West Nile, SARS, Lyme disease and hundreds more that have occurred over the last several decades — don't just happen. They are a result of things people do to nature.

Disease, it turns out, is largely an environmental issue. Sixty percent of emerging infectious diseases that affect humans are zoonotic — they originate in animals. And more than two-thirds of those originate in wildlife.

Teams of veterinarians and conservation biologists are in the midst of a global effort with medical doctors and epidemiologists to understand the "ecology of disease." It is part of a project called Predict, which is financed by the United States Agency for International Development. Experts are trying to figure out, based on how people alter the landscape — with a new farm or road, for example — where the next diseases are likely to spill over into humans and how to spot them when they do emerge, before they can spread. They are gathering blood, saliva and other samples from high-risk wildlife species to create a library of viruses so that if one does infect humans, it can be more quickly identified. And they are studying ways of managing forests, wildlife and livestock to prevent diseases from leaving the woods and becoming the next pandemic.

It isn't only a public health issue, but an economic one. The World Bank has estimated that a severe influenza pandemic, for example, could cost the world economy $3 trillion.

The problem is exacerbated by how livestock are kept in poor countries, which can magnify diseases borne by wild animals. A study released earlier this month by the International Livestock Research Institute found that more than two million people a year are killed by diseases that spread to humans from wild and domestic animals.

The Nipah virus in South Asia, and the closely related Hendra virus in Australia, both in the genus of henipah viruses, are the most urgent examples of how disrupting an ecosystem can cause disease. The viruses originated with flying foxes, Pteropus vampyrus, also known as fruit bats. They are messy eaters, no small matter in this scenario. They often hang upside down, looking like Dracula wrapped tightly in their membranous wings, and eat fruit by masticating the pulp and then spitting out the juices and seeds.

The bats have evolved with henipah over millions of years, and because of this co-evolution, they experience little more from it than the fruit bat equivalent of a cold. But once the virus breaks out of the bats and into species that haven't evolved with it, a horror show can occur, as one did in 1999 in rural Malaysia. It is likely that a bat dropped a piece of chewed fruit into a piggery in a forest. The pigs became infected with the virus, and amplified it, and it jumped to humans. It was startling in its lethality. Out of 276 people infected in Malaysia, 106 died, and many others suffered permanent and crippling neurological disorders. There is no cure or vaccine. Since then there have been 12 smaller outbreaks in South Asia.

In Australia, where four people and dozens of horses have died of Hendra, the scenario was different: suburbanization lured infected bats that were once forest-dwellers into backyards and pastures. If a henipah virus evolves to be transmitted readily through casual contact, the concern is that it could leave the jungle and spread throughout Asia or the world. "Nipah is spilling over, and we are observing these small clusters of cases — and it's a matter of time that the right strain will come along and efficiently spread among people," says Jonathan Epstein, a veterinarian with EcoHealth Alliance, a New York-based organization that studies the ecological causes of disease.

That's why experts say it's critical to understand underlying causes. "Any emerging disease in the last 30 or 40 years has come about as a result of encroachment into wild lands and changes in demography," says Peter Daszak, a disease ecologist and the president of EcoHealth.

Emerging infectious diseases are either new types of pathogens or old ones that have mutated to become novel, as the flu does every year. AIDS, for example, crossed into humans from chimpanzees in the 1920s when bush-meat hunters in Africa killed and butchered them.

Diseases have always come out of the woods and wildlife and found their way into human populations — the plague and malaria are two examples. But emerging diseases have quadrupled in the last half-century, experts

say, largely because of increasing human encroachment into habitat, especially in disease "hot spots" around the globe, mostly in tropical regions. And with modern air travel and a robust market in wildlife trafficking, the potential for a serious outbreak in large population centers is enormous.

The key to forecasting and preventing the next pandemic, experts say, is understanding what they call the "protective effects" of nature intact. In the Amazon, for example, one study showed an increase in deforestation by some 4 percent increased the incidence of malaria by nearly 50 percent, because mosquitoes, which transmit the disease, thrive in the right mix of sunlight and water in recently deforested areas. Developing the forest in the wrong way can be like opening Pandora's box. These are the kinds of connections the new teams are unraveling.

Public health experts have begun to factor ecology into their models. Australia, for example, has just announced a multimillion-dollar effort to understand the ecology of the Hendra virus and bats.

IT'S not just the invasion of intact tropical landscapes that can cause disease. The West Nile virus came to the United States from Africa but spread here because one of its favored hosts is the American robin, which thrives in a world of lawns and agricultural fields. And mosquitoes, which spread the disease, find robins especially appealing. "The virus has had an important impact on human health in the United States because it took advantage of species that do well around people," says Marm Kilpatrick, a biologist at the University of California, Santa Cruz. The pivotal role of the robin in West Nile has earned it the title "super spreader."

Hotspots for emerging diseases

And Lyme disease, the East Coast scourge, is very much a product of human changes to the environment: the reduction and fragmentation of large contiguous forests. Development chased off predators — wolves, foxes, owls and hawks. That has resulted in a fivefold increase in white-footed mice, which are great "reservoirs" for the Lyme bacteria, probably because they have poor immune systems. And they are terrible groomers. When possums or gray squirrels groom, they remove 90 percent of the larval ticks that spread the disease, while mice kill just half. "So mice are producing huge numbers of infected nymphs," says the Lyme disease researcher Richard Ostfeld.

"When we do things in an ecosystem that erode biodiversity - we chop forests into bits or replace habitat with agricultural fields - we tend to get rid of species that serve a protective role," Dr. Ostfeld told me. "There are a few species that are reservoirs and a lot of species that are not. The ones we encourage are the ones that play reservoir roles." Dr. Ostfeld has seen two emerging diseases - babesiosis and anaplasmosis - that affect humans in the ticks he studies, and he has raised the alarm about the possibility of their spread.

The best way to prevent the next outbreak in humans, specialists say, is with what they call the One Health Initiative — a worldwide program, involving more than 600 scientists and other professionals, that advances the idea that human, animal and ecological health are inextricably linked and need to be studied and managed holistically.

"It's not about keeping pristine forest pristine and free of people," says Simon Anthony, a molecular virologist at EcoHealth. "It's learning how to do things sustainably. If you can get a handle on what it is that drives the emergence of a disease, then you can learn to modify environments sustainably."

The scope of the problem is huge and complex. Just an estimated 1 percent of wildlife viruses are known. Another major factor is the immunology of wildlife, a science in its infancy. Raina K. Plowright, a biologist at Pennsylvania State University who studies the ecology of disease, found that outbreaks of the Hendra virus in flying foxes in rural areas were rare but were much higher in urban and suburban animals. She hypothesizes that urbanized bats are sedentary and miss the frequent exposure to the virus they used to get in the wild, which kept the infection at low levels. That means more bats — whether from poor nutrition, loss of habitat or other factors — become infected and shed more of the virus into backyards.

THE fate of the next pandemic may be riding on the work of Predict. EcoHealth and its partners — the University of California at Davis, the Wildlife Conservation Society, the Smithsonian Institution and Global Viral Forecasting — are looking at wildlife-borne viruses across the tropics, building a virus library. Most of the work focuses on primates, rats and bats, which are most likely to carry diseases that affect people.

Most critically, Predict researchers are watching the interface where deadly viruses are known to exist and where people are breaking open the forest, as they are along the new highway from the Atlantic to the Pacific across the Andes in Brazil and Peru. “By mapping encroachment into the forest you can predict where the next disease could emerge,” Dr. Daszak, EcoHealth’s president, says. “So we’re going to the edge of villages, we’re going to places where mines have just opened up, areas where new roads are being built. We are going to talk to people who live within these zones and saying, ‘what you are doing is potentially a risk.’ ”
It might mean talking to people about how they butcher and eat bush meat or to those who are building a feed lot in bat habitat. In Bangladesh, where Nipah broke out several times, the disease was traced to bats that were raiding containers that collected date palm sap, which people drank. The disease source was eliminated by placing bamboo screens (which cost 8 cents each) over the collectors.
EcoHealth also scans luggage and packages at airports, looking for imported wildlife likely to be carrying deadly viruses. And they have a program called PetWatch to warn consumers about exotic pets that are pulled out of the forest in disease hot spots and shipped to market.
All in all, the knowledge gained in the last couple of years about emerging diseases should allow us to sleep a little easier, says Dr. Epstein, the EcoHealth veterinarian. “For the first time,” he said, “there is a coordinated effort in 20 countries to develop an early warning system for emerging zoonotic outbreaks.”
Jim Robbins is a frequent contributor to the Science section of The New York Times.

http://www.scientificamerican.com/article.cfm?id=last-worm-tropical-disease-near-eradication

The World’s Last Worm: A Dreaded Disease Nears Eradication

A dreaded tropical disease is on the verge of eradication

By Roxanne Nelson | Sunday, July 15, 2012 | 17

A parasite that has plagued the human race since antiquity is poised to become the second human disease after smallpox to be eradicated. “We are approaching the demise of the last guinea worm who will ever live on earth,” says former U.S. president Jimmy Carter, whose Carter Center has spearheaded the eradication effort.
Unlike polio's high-profile eradication program, the mission to eliminate guinea worm disease has largely been off the public's radar. Affecting some of the poorest and most remote communities in Africa—97 percent of cases are in South Sudan—guinea worm is a parasitic infection caused by the nematode roundworm Dracunculus medinensis. It is the only disease transmitted solely by drinking water, and humans are its only reservoir, says James Hughes, professor of medicine and public health at Emory University. The disease spreads when villagers consume water containing fleas that harbor guinea worm larvae. The larvae grow to maturity inside the human body and emerge after a year as a fully grown two- to three-foot-long worm that often exits through the leg or foot. It is an excruciatingly painful process, and individuals often immerse the limb in water to cool the burning sensation, which starts the cycle all over again.
Since 1986 groups such as the Carter Center have distributed cloth water filters to villagers and educated residents about how not to spread the infection. They have also selectively used Abate, a larvicide, to control fleas in the drinking water.
So far the efforts have resulted in a 99 percent reduction in infections, says Sharon Roy of the U.S. Centers for Disease Control and Prevention. In 1986 there were 3.5 million cases, as compared with only 1,060 in 2011 and a mere five as of the first few months of 2012. *COMMENT AT ScientificAmerican.com/jul2012*

http://www.bbc.co.uk/news/health-18853597

Aspirin a 'no brainer' against cancer after screening

A mass-screening programme for 50- to 70-year-olds could cut the risk of stomach bleeds due to daily doses of aspirin, cancer experts have said.

By Susan Watts Newsnight science editor, BBC News

About a third of this group carry the bacterium Helicobacter pylori, which makes stomach bleeds three times more likely - and antibiotics eradicate it. Research has shown taking low-doses of aspirin can cut the risk of cancer. Professor Jack Cuzick said screening would make the choice to take it a "no-brainer".
University of London epidemiology Prof Cuzick told the BBC's Newsnight programme: "The test is cheap and very easy to do, and eradication takes only five days. "Bleeding is the only major setback.
"It's trying to identify those who are infected that matters."
The society working with an international team of experts on cancer prevention is expected to publish a statement on the risks and benefits of long-term aspirin use within weeks.
"We will say this looks very important and needs to be further evaluated", Prof Cuzick said.
The society first looked into aspirin as a cancer-prevention measure in 2009, and has reconvened as evidence of potential benefits has grown.

Heart attack

Taking low-dose aspirin for five years halves the risk of developing colon cancer, according to data published two years ago by Peter Rothwell, from Oxford University.

But Prof Cuzick told Newsnight the most up-to-date data showed "much stronger results".

Last year, research indicated daily low-dose aspirin cut the risk of dying by 66% for oesophageal cancer and 25% for lung cancer. When researchers looked at all solid cancers together, the risk also fell, by 25%.

This year, the team looked at aspirin's effect on the spread of cancer, and found it reduced the risk of secondary spread to the lungs, liver and the brain by "about half".

Low-dose aspirin is already recommended to cut the risk of heart attack and stroke, but there are no national guidelines on who should consider taking it to prevent cancer, or how much to take.

You can see more on The Aspirin Debate - with Newsnight's science editor, Susan Watts, on BBC Two at 22:30 BST on Monday.