http://www.eurekalert.org/pub_releases/2011-01/cmc-bip011711.php

Beyond improving Parkinson's symptoms, does deep brain stimulation stall their progression?

Michele Tagliati, M.D., new director of Cedars-Sinai's Movement Disorders Program, says certain disabilities "remain remarkably stable" long-term in treated patients

LOS ANGELES (Jan. 17, 2011) – Parkinson's disease symptoms begin subtly and worsen as damage to certain brain cells continues. But an electrical stimulation device implanted deep in the brain and programmed remotely, along with medications, may provide some control of "motor symptoms" common to the disease, such as shaking, stiffness, and loss of muscle control.

What happens, however, if the drugs are stopped and the device is switched off after five years? Are the symptoms far worse than they were to start, as might be expected with a "progressive" degenerative disorder?

Surprisingly, no, says neurologist Michele Tagliati, M.D., director of the Movement Disorders Program at Cedars-Sinai Medical Center and one of the nation's leading experts in deep brain stimulation therapy.

He and colleagues at Mount Sinai School of Medicine, where he served before joining Cedars-Sinai in September, evaluated several of their deep brain stimulation patients at one-year intervals: 21 patients at year one; 17 at year two; 14 at year three; 16 at year four; and nine at year five.

Part of their analysis – evaluating patients while their drugs temporarily were discontinued but brain stimulation continued – confirmed previous studies: Deep brain stimulation is an effective therapy for advanced Parkinson's disease up to five years from implantation, although there is a gradual reduction in benefit over time. This effectiveness decline usually is attributed to the disease's unrelenting progression.

But the researchers also explored Parkinson's natural progression in these patients by temporarily discontinuing both their drugs and brain stimulation, then comparing motor function at these yearly intervals with pre-treatment scores.

"In these patients who were effectively treated with DBS stimulation, we found that motor symptoms remained remarkably stable over time. There was no significant progression. Now we need to do larger studies to find out why. It may be, as some have suggested, that deep brain stimulation stabilizes the motor progression of the disease, although other studies indicate that Parkinson's disease may just naturally stabilize after several years of progression," said Tagliati, pointing out that "non-motor" symptoms, including depression, dementia and others, currently do not respond to deep brain stimulation and appear to continue to progress.

Tagliati, who leads an educational course on deep brain stimulator programming every year at the American Academy of Neurology meetings, has been studying the procedure for more than a decade, beginning several years before the device was approved as a therapy by the Food and Drug Administration. He and his counterpart at the University of California, Los Angeles, Jeff M. Bronstein, M.D., Ph.D., recently led a panel of international experts in developing a consensus on key issues related to the procedure for Parkinson's disease. Their document, with insight and guidance, was published in Archives of Neurology in October.

"Providing the most effective therapy requires teamwork and the experience and expertise that come from working in a specialized center. It involves placing the device in precisely the right location, programming and fine-tuning the device, properly adjusting medications, studying many patients and outcomes, learning and teaching – all the activities found in an academic center," Tagliati says. "We're looking forward to pursuing innovative research strategies in the near future. Although we know DBS can help many patients with Parkinson's disease, there's much more we need to learn. We can see that stimulation works but we don't really know how it works."

Most patients suffering from Parkinson's disease first are treated with medication to improve levels of dopamine, a chemical lost when certain brain cells are damaged. But if drugs fail to provide adequate symptom control or if patients have unmanageable side effects, deep brain stimulation may be an option.

The device consists of electrical leads implanted in the brain and a stimulator located near the collarbone. The stimulator is programmed with a remote, handheld controller to block abnormal nerve signals that cause uncontrollable muscle activity.

This procedure does not replace drugs but it often allows their dosage to be reduced; the combination provides better muscle and movement control than drugs alone. Motor function improvements range from 27 percent to 72 percent within a year of deep brain stimulation surgery, according to earlier research.

The new study appears in the November issue of the International Journal of Neuroscience. Tagliati receives speaking honoraria and consulting fees from Medtronic Inc., manufacturer of the stimulation device.

http://news.sciencemag.org/scienceinsider/2011/01/quandary-scientists-prefer-readi.html

Quandary: Scientists Prefer Reading Over Publishing 'Open Access' Papers

by Gretchen Vogel

BERLIN—*Scientists love open-access papers as readers, but as authors they are still skeptical, according to a new study of available journals and researchers' attitudes on the topic.*

The E.U.-sponsored Study of Open Access Publishing (dubbed the SOAP project) surveyed 50,000 researchers for their opinions on open-access journals, which make all their papers freely available online and usually charge authors a fee for each published paper. (*Here's an example called Biogeosciences*.)

The study found overwhelming support for the concept, with 89% of respondents saying that open access is beneficial to their field. But that support didn't always translate into action: Although 53% of respondents said they had published at least one open-access article, overall only about 10% of papers are published in open-access journals. "We cannot ignore this gap anymore," says Salvatore Mele, project leader for open access at CERN near Geneva, Switzerland, and a member of the study team.

The study, which released its full results yesterday at a symposium here, found two main reasons researchers don't submit their work to open-access journals.

Almost 40% said that a lack of funding for author fees was a deterrent. And 30% cited a lack of high-quality open-access journals in their field. Clearly, "journal quality and impact factor is most important—not [open access]—when deciding where to submit" for the majority of scientists, says Peter Strickland of the International Union of Crystallography, which publishes the fully open-access Structure Reports Online as well as seven subscription-based journals.

Requiring authors to make sure the results of their work are freely available has had only partial success. Robert Kiley, head of digital services at the Wellcome Trust's Wellcome Library in London, said at the symposium that open-access rates had risen from 12% to 50% since the funder began requiring its grantees to publish in open-access journals or deposit their papers in a freely available repository. Kiley acknowledged, however, that Wellcome had not imposed sanctions on researchers who failed to comply. "We are trying to persuade people rather than punish them," he said.

The study also makes it clear that open-access journals are proliferating, especially among small publishers. The study found that one-third of open-access papers were published by the more than 1600 open-access publishers that publish only a single journal. Several hundred new open-access journals are being launched each year, noted Caroline Sutton of the Open Access Scholarly Publishers Association in The Hague. "It's really difficult to launch a new subscription-based journal, but the open-access fee model is scalable," she says. "As you receive more submissions and publish more papers, you get more fees."

The study also identified 14 "large publishers" that publish either more than 50 journals or more than 1000 articles per year. The group accounts for roughly one-third of open-access publications, the study found. Other large publishers are catching on. Half a dozen large scientific publishers have announced new open-access journals in the past 6 months, notes Mark Patterson, director of publishing at the Public Library of Science (PLoS). All are modeled on PLoS ONE, the publisher's biggest journal (and main revenue generator). The journal publishes papers after an accelerated peer review in which experts check for scientific rigor but not overall importance. Nature Publishing Group is the latest to jump on board: It announced last week that it was accepting submissions for a new rapid-review, all open-access journal, Scientific Reports, due to begin publishing in June.

Preliminary results from the study are, of course, *freely available online*. Mele says the entire data set and the team's analysis, as well as videos of the symposium, will be available next week via the SOAP project Web site.

http://www.latimes.com/health/la-sci-friend-or-foe-20110115,0,763300.story

An equation for friendship

Researchers at Cornell expand on the long-established social psychology theory of structural balance. They find that such balance is achieved through incremental shifts in relationships.

By Eryn Brown, Los Angeles Times

If only they had been there in 1939: Plugging in numbers representing the friendliness between pairs of nations at the outset of World War II, researchers at Cornell University used a computer program to successfully predict which countries joined the Allied Powers and which lined up with the Axis. They got all of the countries right except for Denmark and Portugal.

The group's work, reported last week in the Proceedings of the National Academies of Science, had less to do with history than with a long-established theory in social psychology called "structural balance," which describes how relationships in a social network evolve over time.

"Structural balance is an appealing and powerful theory, but it had a big missing piece," said Jon Kleinberg, a computer science professor who was part of the team. That gap was a way of showing how small shifts in individual relationships result in particular, predictable outcomes in larger alliances. Crunching a lot of numbers, the team hit upon a method to do just that.

About the middle of the 20th century, social psychologists began studying the dynamics of "relationship triangles" involving three people, companies or other entities that are bound together in a network.

They observed that certain combinations of friendly and antagonistic relationships were stable. These included three mutual friends, or two friends who shared a common enemy.

Other relationship triangles were unstable but likely to evolve into stable configurations.

For instance, among a group of three enemies, the two who disliked each other the least tended to team up against the third, resulting in the two-friends-with-a-shared-enemy scenario. As an example, Kleinberg pointed to the aftermath of the 2008 Democratic primary, when former Democratic rivals Barack Obama and Hillary Rodham Clinton teamed up against Republican John McCain.

A triangle containing one person with two friends who don't get along is inherently unstable. As many people know from personal experience, staying friends with a duo of enemies is hard. "You'd like them to reconcile," Kleinberg said, but each of them wants you "to team up against the other one."

In addition to noting the dynamics within triangles, the structural balance theory makes predictions about the larger network. Once all relationship triangles within a network evolved to their balanced states, psychologists observed, the members of that network either would be entirely friendly or would divide into two opposing camps.

The theory was used to help scholars understand shifting allegiances in international relations and to analyze the way companies compete in business. But though it did a good job explaining the "end state" of a stable social network, Kleinberg said, "it left everyone in the dark as to how everyone gets to that state."

The Cornell team's breakthrough was figuring out, through a series of mathematical calculations and computer simulations, a process by which structural balance can be achieved. Their big insight was that shifts between positive relationships and negative ones don't happen at once. Rather, they occur incrementally, with each one affecting — and being affected by — the incrementally shifting nature of the other relationships in the network.

Alliances and animosities don't just turn on and off in a vacuum. Instead, friendships get nudged in a more positive or more negative direction depending on who else is involved. "Everyone is updating their relationships all the time," Kleinberg said.

Previously, mathematicians got stuck trying to simulate how changes in individual relationships led to a globally balanced state, he said. Running computer simulations that took more incremental shifts into account allowed the Cornell team to (mostly) "predict" the Axis-Allies split, which provided validation for their model.

The discovery impressed scientists long baffled by the mechanism behind structural balance.

"It's a very interesting paper," said Daron Acemoglu, an MIT economist who was not involved in the research. "It has potential applicability to a range of situations."

One of those situations could be the study of online networks, Kleinberg said. For example, companies such as Facebook and Twitter want to make sure their networks don't split into polarized camps. Knowing how the predictions of structural balance theory unfold in the real world could help them design their networks in a way that keeps them cohesive. But, he added, "This is abstracted reality; it's not reality. Realistic scenarios are messier."

http://www.livescience.com/animals/plague-of-bats-and-frogs-110114.html

In Deadly Frog and Bat Plagues, Eerie Similarities

By Wynne Parry, LiveScience Senior Writer

Bats and frogs share a common plight. Fungi that appeared from nowhere are wiping out whole species of amphibians and more than a million bats just by attacking the skin.

And both plights may represent new disease paradigms for wildlife, according to researchers.

"It's taken awhile to really come to terms with how a fungus that infects the skin might be killing an animal," said Paul Cryan, a bat ecologist with the U.S. Geological Survey and the lead author of a study that finds echoes of amphibian chytrid-fungi infections in bats. "With bats and white-nose syndrome, I think we've followed in the footsteps of the chytrid researchers in sort of thinking about new possibilities of disease."

Signs of white-nose syndrome were first spotted in a cave near Albany, N.Y., in 2006, and the culprit, a cold-loving soil fungus called Geomyces destructans, was identified about two years later.

It took much longer to identify the frog fungus — a type of water-dwelling chytrid — responsible for the mysterious amphibian declines. Discussion of the declines appears to have begun at the World Congress of Herpetology in 1989, while the fungal infection now blamed, chytridiomycosis, was first described in 1998, and

the fungi that caused it was named and described a year later, according to Jamie Voyles, a postdoctoral researcher at the University of Idaho, who has studied the disease. “We are used to finding pathogens that get into the body, replicate, and basically take over, like viruses or bacteria," said Vance Vredenburg of San Francisco State University, who also studies amphibians. The scientific community caught on much more quickly with white-nose syndrome, a sign that he said gives him hope.

Mysterious skin infections

The problem: Both fungi infect only the skin. But for these creatures, skin is more than just a covering. In amphibians, the skin plays a crucial role in exchanging gases, water and electrolytes, such as sodium, between the animal and its environment. In 2009, researchers led by Voyles reported that the superficial skin infection caused by the chytrid fungus disrupted the transport of electrolytes, leading to cardiac arrest and death among frogs.

Like its partner in crime, the white-nose syndrome fungus attacks the skin, in particular the wings, and bats' wings do more than just flap. In an article published Nov. 11 in the journal BMC Biology, Cryan and his colleagues suggest the fungus may kill bats by interfering with physiological processes that their wings carry out.

Infected wings resemble crumpled tissue paper as the fungus destroys the skin and its blood vessels, muscle fibers, glands and other components, they write. This infection could be causing a host of ultimately fatal problems, including dehydration, which leads bats to awaken during hibernation and deplete their stored fat; obstructed blood flow, which interferes with bats' ability to exchange gases through their wing membrane; the loss of body heat, and impaired flying ability, according to the authors. This disease, they said, could represent a new paradigm for how mammals die from diseases.

"In general, we don't see a lot of life-threatening fungal pathogens," Cryan said. "Athlete's foot doesn't kill us."

Out of the blue?

Both killer fungi also appeared to emerge out of nowhere. Chytrid fungi live in water and were not known to cause infections in vertebrates until one such species, Batrachochytrium dendrobatidis, often called Bd, was fingered for causing amphibian deaths. Meanwhile, G. destructans lives in soil and was first identified in 2008, as scientists sought to figure out what was killing the bats.

This is a bad sign, because it means neither fungi is dependent on its bat or frog hosts for survival, according to Arturo Casadevall, chairman of the department of microbiology and immunology at Albert Einstein College of Medicine in New York. "Measles doesn't kill every human that it infects," Casadevall said, explaining that the measles virus needs to jump from human to human to survive. On the other hand, "G. destructans goes back into the soil. It doesn't care if there are bats around."

It's possible these fungi became killers by accident. While most pathogens have an evolutionary history with their host, like that between the measles virus and humans, the white-nose fungus and the chytrid fungus may have become virulent alone (without a host) and by chance.

For example, G. destructans' natural home, the soil, is a harsh place. In it, microbes face intense competition for nutrients, the threat of being eaten by amoeba, and shifting, extreme environmental conditions. As a result, they survive by adapting quickly, and these adaptations could, by chance, make them harmful to animals they encounter. This phenomenon is called accidental virulence, Casadevall explains in a review published in the journal Eukaryotic Cell in December of 2007.

Rising death tolls

White-nose syndrome has wiped out more than a million bats in North America, according to the U.S. Fish and Wildlife Service, and it is threatening the once-common little brown bat with extinction. However, in Europe, the fungus doesn't appear to kill the bats it infects, a puzzle that could yield important clues.

"The most likely hope these bats will have is individuals will survive the infection, and the populations will evolve resistance," Cryan said. But until this happens, researchers must continue working to understand how the infection kills the bats in the hopes of intervening, he said.

Many amphibians have already been wiped out. In 2007, a study published in the journal EcoHealth suggested that chytrid infections had caused the decline or extinction of up to roughly 200 species of frogs. "The impact of chytridiomycosis on frogs is the most spectacular loss of vertebrate biodiversity due to disease in recorded history," wrote the authors. And historically, it was believed that infectious disease could not cause extinctions, because as the population dropped, disease transmission would also decrease, Voyles said.

"Chytridiomycosis is probably the best empirical example that we know of to demonstrate disease-induced extinction," Voyles told LiveScience in an e-mail. Other examples of devastating disease exist, she wrote, "But in this sense, I think chytridiomycosis has led to a paradigm shift in how we view infectious diseases in wildlife."

http://www.physorg.com/news/2011-01-aan-guideline-plasma-exchange-effective.html

AAN guideline: Plasma exchange effective in treating severe MS relapses, neuropathies

A new guideline from the American Academy of Neurology recommends using plasma exchange to treat people with severe relapses in multiple sclerosis (MS) and related diseases, as well as those with certain kinds of nerve disorders known as neuropathies.

The guideline is published in the January 18, 2011, print issue of Neurology, the medical journal of the American Academy of Neurology.

Plasma exchange, formally known as plasmapheresis, is the process of taking blood out of the body, removing constituents in the blood's plasma thought to be harmful, and then transfusing the rest of the blood (mainly red blood cells) mixed with replacement plasma back into the body.

The guideline recommends doctors consider using plasma exchange as a secondary treatment for severe flares in relapsing forms of MS and related diseases. The treatment was not found to be effective for secondary progressive and chronic progressive forms of MS.

According to the guideline, doctors should offer plasma exchange for treatment of severe forms of Guillain-Barré syndrome and for temporary treatment of chronic inflammatory demyelinating polyneuropathy. Plasma exchange may also be considered for treatment of some other kinds of inflammatory neuropathies.

"These types of neurologic disorders occur when the body's immune system mistakenly causes damage to the nervous system. Plasma exchange helps because it removes factors in the plasma thought to play a role in these disorders," said guideline lead author Irene Cortese, MD, a neurologist with the National Institutes of Health in Bethesda, Md., and a member of the American Academy of Neurology.

The guideline authors also looked at the use of plasma exchange for other neurologic disorders, including myasthenia gravis and pediatric autoimmune neuropsychiatric disorders (PANDAS), but there was not enough evidence to determine whether it is an effective treatment.

Side effects of plasma exchange include infection and blood-clotting issues.

Provided by American Academy of Neurology

http://www.physorg.com/news/2011-01-heart-failure-patients-die-wards.html

Heart failure patients twice as likely to die if admitted to general wards

Heart failure patients admitted to general wards are twice as likely to die as those admitted to cardiology wards, shows a national audit of the treatment of the condition, published online in the journal Heart.

Women fared worse than men when it comes to appropriate investigations and treatment, the findings suggest, although death rates were similar. In 2006/7, heart failure accounted for more than a quarter of a million hospital deaths and discharges in England and Wales, equating to around 2.5 million bed days a year and at an annual cost to the NHS of £563 million.

The authors draw their conclusions from a survey of the first 10 patients admitted each month with a primary diagnosis of heart failure to 86 hospitals across England and Wales between April 2008 and March 2009.

During this period, just over 6,000 patients, with an average age of 78, were admitted with the condition. Almost half of these (43%) were women.

At admission, less than a third (30%) were reported to be breathless at rest and under half (43%) as having swollen feet/ankles. These are both diagnostic features of heart failure.

Appropriate investigations were not always carried out, the survey shows, with those admitted to general medical wards less likely to receive these than those admitted to cardiology wards.

Most patients (75%) were given a heart trace monitor test (echocardiogram). But only two thirds of those (65%) admitted to general medical wards were given this test.

This showed that the left ventricular ejection fraction (LVEF), an indicator of the pump action of one of the two lower chambers of the heart, was 40% or less in most of those admitted.

But LVEF was not recorded in one in four patients. And those with an LVEF of under 40% or in whom LVEF was not recorded were more likely to be women, older, and managed on general medical wards.

Levels of natriuretic peptides, which are a much effective test for heart failure, and a much better barometer of likely outcome than LVEF, say the authors, were only measured in 1% of patients, despite National Institute of Health and Clinical Excellence recommendations.

Half the patients were admitted to cardiology wards. Compared with those managed on general wards, they tended to be younger and were more likely to be men. Those admitted to general medical wards were twice as likely to die as those admitted to cardiology wards, even after taking account of other risk factors.

While most patients, in whom discharge drug treatment was recorded, were given the appropriate medicines, only half were prescribed beta blockers. Men and younger patients were more likely to be given these drugs.

"Currently, hospital provision of care is suboptimal and the outcome of patents poor. The same rules that apply to suspected cancer should pertain to a disease with such a malign prognosis as heart failure," conclude the authors.

This means ready availability of natriuretic peptide testing, prompt referral to a specialist and appropriately trained staff to manage the condition during and after hospital admission, they say.

Provided by British Medical Journal

http://www.eurekalert.org/pub_releases/2011-01/uonc-nmc011811.php

New molecule could save brain cells from neurodegeneration, stroke

CHAPEL HILL, N.C. – *Researchers at the University of North Carolina at Chapel Hill have discovered a molecule that can make brain cells resistant to programmed cell death or apoptosis.*

This molecule, a tiny strand of nucleotides called microRNA-29 or miR-29, has already been shown to be in short supply in certain neurodegenerative illnesses such as Alzheimer's disease and Huntington's disease. Thus, the discovery could herald a new treatment to prompt brain cells to survive in the wake of neurodegeneration or acute injury like stroke.

"There is the real possibility that this molecule could be used to block the cascade of events known as apoptosis that eventually causes brain cells to break down and die," said senior study author Mohanish Deshmukh, PhD, associate professor of cell and developmental biology.

The study, published online Jan. 18, 2011, in the journal Genes & Development, is the first to find a mammalian microRNA capable of stopping neuronal apoptosis.

Remarkably, a large number of the neurons we are born with end up dying during the normal development of our bodies. Our nerve cells must span great distances to ultimately innervate our limbs, muscles and vital organs. Because not all nerve cells manage to reach their target tissues, the body overcompensates by sending out twice as many neurons as required. The first ones to reach their target get the prize, a cocktail of factors needed for them to survive, while the ones left behind die off. Once that brutal developmental phase is over, the remaining neurons become impervious to apoptosis and live long term.

But exactly what happens to suddenly keep these cells from dying has been a mystery. Deshmukh thought the key might lie in microRNAs, tiny but powerful molecules that silence the activity of as many as two-thirds of all human genes. Though microRNAs have been a hotbed of research in recent years, there have been relatively few studies showing that they play a role in apoptosis. So Deshmukh and his colleagues decided to look at all of the known microRNAs and see if there were any differences in young mouse neurons versus mature mouse neurons.

One microRNA jumped out at them, an entity called miR-29, which at that time had never before been implicated in preventing apoptosis. When the researchers injected their new molecule into young neurons, which are able to die if instructed, they found that the cells became resistant to apoptosis, even in the face of multiple death signals.

They then decided to pinpoint where exactly this molecule played a role in the series of biochemical events leading to cell death. The researchers looked at a number of steps in apoptosis and found that miR-29 acts at a key point in the initiation of apoptosis by interacting with a group of genes called the BH3-only family. Interestingly, the microRNA appears to interact with not just one but as many as five members of that family, circumventing a redundancy that existed to allow cell death to continue even if one of them had been blocked.

"People in the field have been perplexed that when they have knocked-out any one of these members it hasn't had a remarkable effect on apoptosis because there are others that can step in and do the job," said Deshmukh. "The fact that this microRNA can target multiple members of this family is very interesting because it shows how a single molecule can basically in one stroke keep apoptosis from happening. Interestingly, it only targets the members that are important for neuronal apoptosis, so it may be a way of specifically preserving cells in the brain without allowing them to grow out of control (and cause cancer) elsewhere in the body."

Deshmukh is currently developing mouse models where miR-29 is either "knocked-out" or overactive and plans to cross them with models of Alzheimer's disease, Parkinson's disease and ALS to see if it can prevent neurodegeneration. He is also actively screening for small molecule compounds that can elevate this microRNA and promote neuronal survival.

The research was funded by the National Institutes of Health. Study co-authors were Adam J. Kole, a graduate student in Deshmukh's lab; Vijay Swahari, research technician; and Scott M. Hammond, PhD, associate professor of cell and developmental biology.

http://www.eurekalert.org/pub_releases/2011-01/wios-wis011811.php

Weizmann Institute scientists discover: Antioxidants cause fertility problems in females

Antioxidants are sold over the counter everywhere. They're added to food, drink and face cream. But according to Prof. Nava Dekel of the Biological Regulation Department, we still don't have a complete understanding of how they act in our bodies.

New research by Dekel and her team, recently published in the Proceedings of the National Academy of Sciences USA (PNAS), has revealed a possible unexpected side effect of antioxidants: They might cause fertility problems in females.

Common antioxidants include vitamins C and E. These work by eliminating molecules called reactive oxygen species that are produced naturally in the body. Stress can cause these chemically active molecules to be overproduced; in large amounts they damage cells indiscriminately. By neutralizing these potentially harmful substances, antioxidants may, theoretically, improve health and slow down the aging process.

But when Dekel and her research team including her former and present Ph.D. students Dr. Ketty Shkolnik and Ari Tadmor applied antioxidants to the ovaries of female mice, the results were surprising: ovulation levels dropped precipitously. That is, very few eggs were released from the ovarian follicles to reach the site of fertilization, compared to those in untreated ovaries.

To understand what lies behind these initial findings, the team asked whether it is possible that the process of ovulation might rely on the very 'harmful' substances destroyed by antioxidants – reactive oxygen species.

Further testing in mice showed that this is, indeed, the case. In one experiment, for instance, Dekel and her team treated some ovarian follicles with luteinizing hormone, the physiological trigger for ovulation, and others with hydrogen peroxide, a reactive oxygen species. The results showed hydrogen peroxide fully mimicked the effect of the ovulation-inducing hormone. This implies that reactive oxygen species that are produced in response to luteinizing hormone serve, in turn, as mediators for this physiological stimulus leading to ovulation.

Among other things, these results help fill in a picture that has begun to emerge in recent years of fertility and conception, in which it appears that these processes share a number of common mechanisms with inflammation. It makes sense, says Dekel, that substances which prevent inflammation in other parts of the body might also get in the way of normal ovulation, and so more caution should be taken when administering such substances.

Much of Dekel's research has focused on fertility -- her previous results are already helping some women become pregnant. Ironically, the new study has implications for those seeking the opposite effect. Dekel: 'On the one hand, these findings could prove useful to women who are having trouble getting pregnant. On the other, further studies might show that certain antioxidants might be effective means of birth control that could be safer than today's hormone-based prevention.'

Dekel and her team are now planning further studies to investigate the exact mechanics of this step in ovulation and to examine its effect on mice when administered in either food or drink. In addition, they plan to collect data on the possible link between females being administered antioxidant supplements and the difficulty to conceive.

Prof. Nava Dekel's research is supported by the M.D. Moross Institute for Cancer Research; the Jeanne and Joseph Nissim Foundation for Life Sciences Research; the Yeda-Sela Center for Basic Research; the Willner Family Center for Vascular Biology – Head; the Dwek Family Biomedical Research Fund; the Phyllis and Joseph Gurwin Fund for Scientific Advancement; and the J & R Foundation. Prof. Dekel is the incumbent of the Philip M. Klutznick Professorial Chair of Developmental Biology

http://www.eurekalert.org/pub_releases/2011-01/w-ami011411.php

Antioxidants may improve chances of conceiving in male subfertility

Antioxidant supplements may benefit couples who have difficulty conceiving naturally, according to a new systematic review published today in The Cochrane Library.

The review provides evidence from a small number of trials that suggest the partners of men who take antioxidants are more likely to become pregnant.

Male subfertility affects one in 20 men. Chemicals called reactive oxygen species (ROS) are said to cause damage to cells, and in particular sperm cells, which may result in lowered sperm counts and interfere with their ability to fertilise eggs. Antioxidants include natural and synthetic chemicals, including certain vitamins and minerals, which help to reduce the damage caused by ROS.

The review focused on 34 trials involving 2,876 couples undergoing assisted reproductive techniques such as in vitro fertilisation and sperm injections. Most men in the trials had low sperm counts or low sperm motility. The trials explored the use of many different types of oral antioxidants, including vitamin E, L-carnitine, zinc and magnesium.

Compared to controls, a couple was more likely to have a pregnancy or live birth if the man took antioxidants. However, these results are based on just 964 of the couples in the review for pregnancies and 214 couples for live births. Other trials tested the effects of antioxidants on sperm motility and concentration and showed mostly positive effects, although study group sizes were small.

"When trying to conceive as part of an assisted reproductive program, it may be advisable to encourage men to take oral antioxidant supplements to improve their partners' chances of becoming pregnant," said lead researcher Marian Showell, who works in Obstetrics and Gynaecology at the University of Auckland in Auckland, New Zealand. "However, these conclusions are currently based on limited evidence."

There were not enough data comparing different antioxidants to reach any conclusions about the relative effectiveness of supplements. "We need more head-to-comparisons to understand whether any one antioxidant is performing better than any other," said Showell.

http://www.eurekalert.org/pub_releases/2011-01/w-dut011411.php

Drug used to treat heavy periods will stop trauma patients bleeding to death

Tranexamic acid (TXA), a drug used to treat heavy menstrual periods, could save the lives of tens of thousands of bleeding accident victims each year and reduce combat deaths, say Cochrane researchers.

The researchers carried out a systematic review of trials examining the effectiveness of tranexamic acid (TXA) in patients with bleeding after severe injury.

TXA is an inexpensive drug that reduces clot breakdown. It has been used for many years to reduce heavy menstrual bleeding and is often given during planned surgery to reduce the need for blood transfusion. However, more recently, tranexamic acid has been tested in bleeding trauma patients. Of such patients who die in hospital, nearly half die due to excessive blood loss and most others die from injuries that are worsened by bleeding.

According to the results of the new review, TXA reduces the risk of death in injured patients with severe bleeding by about 10% compared to giving no treatment, which equates to saving over 70,000 lives each year if TXA was used worldwide. The results are based on one large trial involving 20,211 patients and one small trial in 240 patients, both carried out since an earlier, inconclusive review in 2004.

Lead researcher Ian Roberts of the London School of Hygiene & Tropical Medicine in London, UK, said, "TXA reduces the risk of a patient bleeding to death following an injury and appears to have few side effects. It could save lives in both civilian and military settings."

"These results are based on a large number of patients (men and women) who came from many different countries. Given the high quality of the evidence for the benefits of this drug, we recommend it be used more widely in injury victims with bleeding." A separate Cochrane Systematic Review focused on trials of TXA and other similar drugs in people scheduled for non-urgent surgery showed that TXA was highly effective at reducing blood loss and the need for red blood cell transfusion.

http://www.eurekalert.org/pub_releases/2011-01/mcow-nct011811.php

New CPR technique for out-of-hospital cardiac arrest increases survival by 53 percent

A study led by Dr. Tom P. Aufderheide, professor of emergency medicine at The Medical College of Wisconsin, shows an alternative method of cardio-pulmonary resuscitation increases long-term survival of patients.

The study, which is published in the January 19th, 2011 online version of Lancet, and will be in an upcoming publication of Lancet, determined that active compression-decompression cardio-pulmonary resuscitation (CPR) with augmentation of negative intrathoracic pressure gave patients a better chance of survival. When the pressure inside the thorax decreases, blood flow to the heart and brain increases.

About 800,000 people in the U.S., Canada and Europe have an out-of-hospital cardiac arrest every year. The survival rate averages just 5%, in part because standard CPR is inefficient, providing just 25% of healthy blood flow to the heart and brain.

In the randomized study, 46 emergency medical service (EMS) agencies in urban, suburban and rural areas of the USA, including EMS in Oshkosh, provided either standard CPR or the new technique to adults who had a non-traumatic arrest presumed cardiac in nature.

The new technique uses two devices simultaneously to increase circulation. One is a handheld device that attaches with a small suction cup to the patient's chest. After each compression, the suction cup allows the chest to be lifted up, stimulating blood flow. The second device, called an impedance threshold device, attaches to the patient's airway using a facial mask or breathing tube. When the chest lifts upward, the impedance threshold device prevents air from rushing into the lungs. That creates a vacuum inside the chest and helps refill the heart

after each compression. Researchers found in each compression-decompression cycle, the heart and brain receive nearly three times more blood flow when compared with standard CPR.

A total of 813 standard CPR patients and 840 intervention patients were analyzed in the study. Researchers found 6% of the standards CPR patients survived to hospital discharge with favorable neurologic function. That compares with 9% in the intervention group (improvement of survival chance 53% in intervention group). The same proportions of patients in each group survived to one year.

"Based on our findings, active compression-decompression CPR with augmentation of negative intrathoracic pressure should be considered as an alternative to standard CPR to increase long-term survival after cardiac arrest," said Dr. Aufderheide.

Co-authors on the Lancet paper include Dr. Ralph J. Frascone, MD, Department of Emergency Medicine, Regions Hospital, St. Paul, MN; Dr. Marvin A. Wayne, MD, Whatcom County Emergency Services, Department of Emergency Medicine, Peace Health, St. Joseph Medical Center, Bellingham, WA; Dr. Brian D. Mahoney, MD and Dr. Keith G. Lurie, MD, Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN; Dr. Robert A. Swor, DO, Department of Emergency Medicine, William Beaumont Hospital, Royal Oak, MI; Dr. Robert M Domeier, Department of Emergency Medicine, St. Joseph Mercy Hospital, Ann Arbor, MI; Dr. Michael L. Olinger, MD, Department of Emergency Medicine, Indiana University School of Medicine, Indianapolis, IN; Dr. Richard G. Holcomb, PhD, Quintiles Consulting, Rockville, MD; Dr. David Tupper, PhD and Dr. Demetris Yannopoulos, MD, Departments of Neurology and Medicine—Cardiovascular Division, University of Minnesota Medical Center, Minneapolis, MN.

http://www.nytimes.com/2011/01/18/health/research/18regimens.html

Regimens: Doubt on IV Fluids as Routine for Trauma Victims

By NICHOLAS BAKALAR

Giving intravenous fluids before taking an accident victim to the hospital is routine in many emergency services systems, but a new study suggests that the practice may do more harm than good.

Researchers analyzed a national database covering the years 2001 to 2005, which included complete records for 311,071 patients with penetrating or blunt injuries. Their study is being published in The Annals of Surgery.

In almost every way they analyzed the data — by blunt or penetrating injury, by gunshot wound, by low blood pressure of the victim, by immediate surgery — giving IV fluids was associated with higher rates of death. For some types of injuries the differences were stark. Those with severe head injuries were 34 percent more likely to die if they came to the hospital on IV fluids; those with a gunshot wound and low blood pressure were more than 70 percent more likely to die.

The researchers suggest that inserting an IV can mean critical delays in getting to the hospital, and that giving fluids can increase bleeding or release a blood clot that has already formed.

“It’s hard to say it, but the new intervention is to do less,” Dr. Elliott R. Haut, an associate professor of surgery at Johns Hopkins and the lead author of the study. “The best thing to do is rapidly bring the patient to a trauma center.”

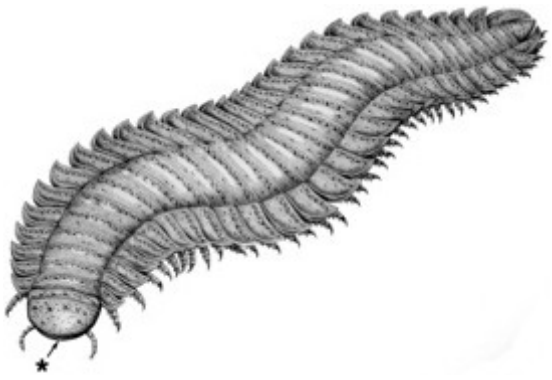
http://blogs.nationalgeographic.com/blogs/news/chiefeditor/2011/01/largest-landdwelling-bug-of-al.html

Largest Land-Dwelling "Bug" of All Time

The giant extinct invertebrate Arthropleura resembled some modern millipedes, but could grow to be more than one-and-a-half feet wide, and may sometimes have been more than six feet long.

By Hans-Dieter Sues

As a kid I enjoyed watching those old sci-fi movies (like the 1954 classic Them!) where giant ants and spiders, created by fallout from atomic explosions, laid waste to cities and towns. While most of us are not overly fond of "creepy-crawlies" invading our homes, many people love the frisson of learning that there were once really big animals like these.



Fortunately, the laws of nature impose tight limits on the maximum size that arthropods can attain. The arthropod body is completely encased in an exoskeleton. The legs are made up of jointed tubes that contain the muscles necessary for their movement. As the animal's size increases, the walls of these leg tubes rapidly increase in thickness, and operating the limbs soon would be impossible if the animal grew too big.

Reconstruction of the giant millipede Arthropleura from the Pennsylvanian and earliest Permian of North America and Europe. The head capsule (marked by an asterisk) is shown tucked under the first plate (collum), as in present-day millipedes. **Courtesy and copyright of Dr. Elke Gröning (Technische Universität Clausthal-Zellerfeld).**

Another constraint faced by large arthropods is breathing. Small forms such as insects can breathe through tubes (tracheae) that open on the outside of the body. The body then absorbs the oxygen into the haemolymph (blood) through specialized soft membranes. The surface area of a body increases in proportion to the square of its dimensions, but the body's volume increases as the cube. Thus if the size of an animal doubles, its body volume (which needs to be supplied by oxygen) increases eightfold. This geometrical relationship significantly constrains size increase. Thus, no monster bugs will ever menace humanity!

During the Pennsylvanian and earliest Permian periods (about 320 to 290 million years before present), much of present-day North America and Europe was located close to the equator and was covered by vast, richly vegetated swamps. The remains of this vegetation ultimately formed the great coal deposits that fuelled the Industrial Revolution and to this day remain a key energy resource. These ancient swamps were home to many large arthropods including early dragonfly relatives with wingspans in excess of two feet and the subject of this blog, the giant millipede Arthropleura. One species of Arthropleura ("jointed rib") is the largest known land-dwelling invertebrate of all time.

The flattened body of Arthropleura is composed of approximately 30 jointed segments, each of which was covered by two side plates and one center plate. The ratio of pairs of legs to body segments was approximately 8:6, similar to some present-day millipedes. Typically, the body armor of Arthropleura fell apart after the death of the animal, and only individual segments or plates were preserved as fossils.

Unfortunately, nobody has yet found a complete large individual of Arthropleura. One partial body fossil from southwestern Germany has a length of 90 cm (3 ft.). A trackway ascribed to a large Arthropleura on a Pennsylvanian-age sandstone surface from Nova Scotia (Canada) comprises two parallel rows of small imprints and is 50 cm (19.7 in.) wide. It is estimated that the maker of this track was at least 1.7 m (5.6 ft.) long. Similar trackways have also been discovered in the United States and in Scotland. The size of some isolated armor segments indicates that Arthropleura adults could attain a length of at least 2 m (6.6 ft.). The only even larger arthropod was the aquatic Early Devonian "sea scorpion" Jaekelopterus, which, based on one isolated chelicera (pincer-like mouth part), reached an estimated length of 2.5 m (8.2 ft.).

As no complete fossils of large Arthropleura are known, the interpretation of their structure has been difficult. In the last few years, two German researchers--Otto Kraus, an expert on present-day millipedes, and Carsten Brauckmann, a specialist on ancient arthropods--have undertaken a detailed re-examination of the known fossils. Many older reconstructions of Arthropleura showed a large rounded "head end," but this appears to be the first armor plate, known as the collum, and the actual head capsule was tucked under the collum, as it is in present-day millipedes. Another interesting result of the new research is the discovery that the sturdy-looking body armor is only a few millimeters thick and was not reinforced by calcium carbonate (as, for example, in crustaceans). Considering their size, adult Arthropleura would have had few if any enemies in the Pennsylvanian coal swamps and therefore no need for heavy armor.

How did Arthropleura breathe? There are no traces of a tracheal system, and gas exchange through the body surface would have been insufficient for the oxygen needs of such a large animal. There are paired, pocket-like features on the underside of each body segment, and these pockets have a peculiar granulated surface. It has been suggested that a thin layer of air covered these surfaces and oxygen could be absorbed by diffusion through them. Geochemical modeling by Robert Berner (Yale University) suggests that the oxygen content of Earth's atmosphere was much higher during Pennsylvanian times (30 to 35%) than today (21% free oxygen), so large arthropods could have breathed more easily than they would have today.

What did Arthropleura eat? An earlier study reported possible gut contents in a specimen from Scotland. These contents were composed of debris from the tree-like club mosses (lycophytes) that formed a major component of the coal swamp vegetation. Restudy of the fossil in question by Kraus, however, indicates that this is an accidental association of a shed skin of an Arthropleura with some plant fragments. Kraus believes that Arthropleura indeed fed on plants but thinks that the enormous quantities of spores shed by swamp plants including lycophytes as well as early growth stages of these plants would have been rich sources of food. Most present-day millipedes feed on dead plant matter, and it is reasonable to assume that Arthropleura did likewise.

The extinction of Arthropleura is probably related to the climatic changes during the Permian Period when increasingly drier conditions led to the disappearance of the coal swamps. The work by Kraus and Brauckmann and other researchers indicates that Arthropleura may be most closely related to the present-day Penicillata, a group including the tiny bristle millipedes (Polyxenus), which are widespread in drier habitats in eastern North America. What a relief that we no longer have to worry about tripping over six-foot millipedes on our hikes through the forest!

http://www.eurekalert.org/pub_releases/2011-01/idso-ssp011411.php

Study suggests possible new treatment for severe 2009 H1N1 infection

Convalescent plasma therapy—using plasma from patients who have recovered from an infection to treat those with the same infection—has been used to treat multiple diseases

However, the efficacy of this treatment in patients with severe 2009 H1N1 influenza is unknown. A study published in the February 15 issue of Clinical Infectious Diseases suggests that convalescent plasma may reduce the death rate in patients severely ill with this type of influenza. (Please see below for a link to the embargoed study online.)

From September 2009 through June 2010, patients from a hospital cluster in Hong Kong with severe 2009 H1N1 infection requiring intensive care were recruited for the study. Of the 93 patients in the study, 20 received the plasma treatment. Prior patients who had recovered from H1N1 infection provided the convalescent plasma for the study. The 73 members of the study who declined the treatment were the study controls.

Mortality in the treatment group was 20 percent, compared to 55 percent in the non-treatment group. The viral load in the treatment group also decreased at a higher rate than in the control group. None of the patients developed adverse events from the treatment.

"One of the benefits of convalescent plasma treatment in patients with severe influenza A infection is that it does not suffer from the problem of drug resistance," said study author Kowk-Yung Yuen, MD, of the University of Hong Kong in China. "Additionally, it would remain effective until the virus has changed significantly enough to affect immunity. This form of treatment may be useful in future novel viral infections."

NOTE*: The study is available online. It is embargoed until 12:01 a.m. EST on Wednesday, Jan. 19, 2011:*

"Convalescent Plasma Treatment Reduced Mortality in Patients With Severe Pandemic Influenza A (H1N1) 2009 Virus Infection" http://www.oxfordjournals.org/our_journals/cid/ciq106.pdf

http://www.newscientist.com/article/mg20927962.900-slime-moulds-bet-the-farm-on-survival.html

Slime moulds bet the farm on survival

*** 19 January 2011 by Bob Holmes**

SLIME moulds have added another skill to their impressive resumé: they practise a primitive form of farming.

Slime moulds - or social amoebas, as biologists now prefer to call them - have been shown to find the shortest route through mazes and pick the most nutritious food from a buffet. These are impressive feats for an organism that lives most of its life as single cells, grazing on bacteria. When food is scarce, the amoebas clump together to migrate to better feeding grounds, and reproduce by forming a capsule full of spores.

While working with several wild strains of the social amoeba Dictyostelium discoideum, Debra Brock of Rice University in Houston, Texas, noticed that a third of the strains always packaged bacteria along with their spores in the capsule. This means they can "seed" a food crop when they colonise a new habitat. These strains - which Brock calls "farmers" - even stop feeding before all the bacteria are gone, to ensure there are some left to store as seed. In contrast, the "non-farmers" keep feeding to the bitter end, leaving no bacteria to package.

To see whether the farmers benefit from the practice, Brock and her colleagues sowed spores from farming and non-farming amoebas onto sterile Petri dishes. Sure enough, the farmers' stored crop gave them a head start, and they outgrew non-farmers. Farmers even beat non-farmers on Petri dishes inoculated with natural soil bacteria, which suggests that the bacteria they store are better food (Nature, DOI: 10.1038/ nature09668). "It's the same as humans," says Brock. "We grow crops, but we aren't going to go out and eat the leaves that we find naturally."

With no evidence that the amoebas weed or fertilise their crop, the interaction might be better termed husbandry rather than farming, says Jacobus Boomsma at the University of Copenhagen, Denmark. Nevertheless, its presence in some strains and not others will let us study the evolutionary origins of the behaviour.

http://www.eurekalert.org/pub_releases/2011-01/icl-cct012011.php

Contagious cancer thrives in dogs by adopting host's genes

A curious contagious cancer, found in dogs, wolves and coyotes, can repair its own genetic mutations by adopting genes from its host animal, according to a new study in the journal Science.

Scientists at Imperial College London have uncovered an unusual process that helps the cancer survive by stealing tiny DNA-containing 'powerhouses' (known as mitochondria) from the cells of the infected animal, to incorporate as its own. They say this may be because genes in the tumour's own mitochondria have a tendency to mutate and degenerate. The results are surprising because mitochondria and their genes are usually only passed from a mother to her offspring.

The findings may have broad implications for halting the spread of similar diseases in other animals and for understanding cancer progression across species. Mitochondrial transfer between genetically distinct cells has previously been observed in the laboratory, but this is the first time it has been demonstrated to occur in nature.

Canine Transmissible Venereal Tumour or CTVT is a very unusual form of cancer that is typically transmitted by mating, though it can also be spread by licking, biting or sniffing tumour-affected areas. The cancer cells themselves move directly from dog to dog, acting like a parasite on each infected animal. Found in most canine breeds throughout the world, the scientists think CTVT is very similar to the transmissible but more fatal cancer seen in the endangered Tasmanian devils of Australia.

Dr Clare Rebbeck, formerly a PhD student on the project at Imperial College London, now working at Cold Spring Harbor Laboratory in the USA, originally set out to explore how the cancers found in different parts of the world were related to one another, using computer analysis of DNA samples. However, her analysis showed that the pattern of relationships for the mitochondrial genes was different to that of the nuclear DNA. In cases the cancers were even more closely related to some dogs than to other some cancers. This finding indicated that the cancers sometimes acquired mitochondria from their hosts.

Professor Austin Burt from the Department of Life Sciences at Imperial College London, who led the research, said: "Our study has revealed that this type of cancer works in a really unexpected way. It raises some really important questions about the progression of other cancers, such as how they repair their own DNA."

The researchers believe that the cancer does not take up new mitochondria with every new host, rather that this functions as an occasional repair mechanism to replace faulty mitochondria. A naturally high rate of genetic mutation in cancers regularly leads to non-functional genes in the CTVT mitochondria, which causes them to lose productivity.

In an earlier study, Imperial's scientists estimated that the earliest CTVT tumour originated from an ancient dog or wolf approximately 10,000 years ago, perhaps when dogs were first domesticated through intensive inbreeding of the more social wolves. Today's results suggest that over this time, the cancer must have evolved the unusual ability to capture mitochondria from its host animal.

The scientists hope their work can be built upon by medical researchers to advance our knowledge of cancer progression in humans and other animal species.

This work has been supported by the Natural Environment Research Council (NERC) and Breakthrough Breast Cancer Research Centre, part of the Institute of Cancer Research (ICR).

http://www.eurekalert.org/pub_releases/2011-01/sri-srs012011.php

Scripps Research scientists find measles' natural nemesis

Cells infected by measles virus pull out a heavy weapon in the form of the enzyme ADAR1

LA JOLLA, CA – Scientists at The Scripps Research Institute have found that a known enzyme in cells protects against measles virus, likely by altering the virus's genetic material, RNA. Cells lacking the enzyme become highly vulnerable to the virus's destructive effects. The enzyme also protects against several other respiratory viruses, including influenza A.

"We believe that host cells use this RNA-editing enzyme to slow these viruses' ability to replicate," said Michael B. A. Oldstone, the study's senior author and a professor at Scripps Research's La Jolla, California campus. The study's first authors are Simone V. Ward, a senior research associate in the Scripps Research Oldstone laboratory, and Cyril X. George of the University of California, Santa Barbara.

The finding represents a significant improvement in the understanding of measles infections, which still kill about 150,000 children and adults around the world every year. The paper, which was published recently in Proceedings of the National Academy of Sciences, has prompted commentaries in the journals Nature Reviews Microbiology and Viruses.

The focus of the study was the enzyme ADAR1 ("adenosine deaminase acting on RNA, 1"), which is known to be produced in high amounts in measles-infected cells. ADAR1 has been suspected as a "restriction factor" that inhibits viral replication.

ADAR1's role against measles has been difficult to nail down, however. In mice genetically engineered to be infectable by measles – a virus that normally infects only humans – ADAR1 is required for embryonic development, as in all mice. Thus the standard "gene knockout" technique, which would enable scientists to see how measles infections proceed without ADAR1, hasn't been feasible.

In this study, Ward, George, and Oldstone, and their colleagues knocked out only one of the two forms of ADAR1 produced in cells. This form, p150, is the one produced in response to infections. For reasons that still aren't clear, mouse embryos cannot grow for long without p150, so the researchers used a standard technique to "immortalize" these p150-knockout embryonic cells—ensuring their continuous supply—and in this way created a useful cell model.

When infected by measles virus, the p150-knockout cells succumbed quickly compared to immortalized control cells that produced p150 normally. "When I looked at the cells only 21 hours after infection, the p150-knockout cells already showed the signs of cell damage typical of measles infection," said Ward. "But the control cells looked exactly like uninfected cells."

In further tests, Ward found p150 also provided significant protection for cells against Newcastle disease virus, Sendai virus, canine distemper virus, and influenza A virus – which are all respiratory viruses like measles, and all members of the paramyxovirus or orthomyxovirus families.

Nine years ago, it was reported that a cellular enzyme known as APOBEC3G protects cells from DNA-based viruses such as HIV, by mutating viral genes. "We're now showing that an analogous gene-editing enzyme also seems to exist for RNA viruses," said Oldstone. With the new cell model, and advanced "conditional knockout" techniques that allow genes to be disrupted in specific organs in adult mice, Ward, Oldstone, and their colleagues now hope to study ADAR1-p150's role in more detail.

One key issue to be resolved is the enzyme's role during brain infections. Measles virus usually results in a relatively mild illness lasting only a week or two, but in rare cases it spreads to the brain and becomes a persistent, always fatal infection known as subacute sclerosing panencephalitis (SSPE). In such cases, the virus doesn't have to spread via cell-to-cell contact, thus exposing itself to the immune system; it can spread more stealthily along the axons and dendrites that connect neurons.

"What we hope to show with our ongoing work is that host neurons are using ADAR1 to slow down this process, turning it into a gradual neurological disease," said Oldstone. ADAR1 might also be exacerbating the neurological symptoms of SSPE, he adds, because its enzymatic activity is known to affect the production of the important neurotransmitter receptors for serotonin and glutamate: "It's an enzyme that has multiple roles," Oldstone concluded.

In addition to Ward and Oldstone, authors of the paper, "RNA editing enzyme adenosine deaminase is a restriction factor for controlling measles virus replication that also is required for embryogenesis," are, Megan J. Welch, Li-Ying Liou, Juan C. de la Torre, and Hanna Lewicki of Scripps Research; Bumsuk Hahm of Scripps Research and the University of Missouri; and Cyril X. George and Charles E. Samuel of the University of California, Santa Barbara. For more information, see http://www.pnas.org/content/108/1/331.abstract

The work was supported by grants from the National Institutes of Health.

http://www.eurekalert.org/pub_releases/2011-01/s-ba012011.php

Breastfeeding -- added protection for cancer survivors?

Review suggests that breastfeeding can partially offset the long-term side effects of cancer treatment

Women who have survived childhood cancer should be advised to breastfeed if they can, in order to offset some of the negative health effects of their earlier cancer treatment. According to Susan Ogg and colleagues from St. Jude Children's Research Hospital in Memphis, Tennessee, making women aware of the benefits of breastfeeding should be part of routine post-cancer diet and healthy lifestyle recommendations. Their work is published online in Springer's Journal of Cancer Survivorship.

It is estimated that one in every 640 young adults between the ages of 20 and 39 will be a survivor of childhood cancer, largely due to the progress in cancer therapy. Specifically, 80 percent of children and adolescents treated with modern cancer therapies now survive. This growing number of cancer survivors faces significant health challenges, including a variety of adverse effects of the cancer itself and its treatment. These late effects include impaired growth and development, organ dysfunction, reproductive difficulties as well as increased risk of cancer re-occurrence.

It is well established that breastfeeding confers a number of health benefits to both infants and their mothers. Ogg and team looked at whether breastfeeding might result in the same benefits to women who have survived childhood cancer.

They reviewed existing research looking at whether women can successfully breastfeed after cancer treatment in childhood, the long-term effects of early cancer treatment on women's health in general and how breastfeeding may help to reduce both the risk and impact of cancer-related toxicity in those who survive. They found that breastfeeding had the potential to influence positively bone mineral density, metabolic syndrome risk factors, cardiovascular disease and secondary tumors - conditions negatively affected by childhood cancer.

Ogg and colleagues conclude: "Alongside advice to eat plenty of fruit and vegetables, abstain from smoking, use suitable sun protection, practice safe sex and take part in regular physical activity, women who have survived childhood cancer and are physically able to breastfeed, should be actively encouraged to do so to help protect them against the many lasting effects of cancer treatment."

References Ogg SW et al (2011). Protective effects of breastfeeding for mothers surviving childhood cancer. Journal of Cancer Survivorship. DOI 10.1007/s11764-010-0169-z

http://www.bbc.co.uk/news/health-12228027

Brenda Jensen's voice restored after larynx transplant

By James Gallagher Health reporter, BBC news

A woman in the US is able to speak for the first time in 11 years after a pioneering voicebox transplant.

Brenda Jensen said the operation, which took place in California, was a miracle which had restored her life.

Thirteen days after the surgery she said her first words: "Good morning, I want to go home." It is the first time a voicebox and windpipe have been transplanted at the same time and only the second time a voice box has ever been transplanted.

Ms Jensen, 52, had been unable to speak on her own since her voicebox was damaged during surgery in 1999.

The tube used to keep her airways open injured her throat and scar tissue stopped her breathing.

Since then, she has been unable to taste or smell food, could breathe only through a hole in her windpipe and could talk only with the help of an electronic voice box.

Pioneering surgery

In October, surgeons at the University of California Davis Medical Centre removed the larynx (voicebox), thyroid gland and 6cm of the trachea (windpipe) from a donor body.

In an 18-hour operation this was transplanted into Ms Jensen's throat and the team connected it to her blood supply and nerves. Thirteen days later she was able to speak her first croaky words and is now able to talk easily for long periods of time.

Ms Jensen said: "This operation has restored my life. I feel so blessed to have been given this opportunity.

"It is a miracle, I'm talking, talking, talking, which just amazes my friends and family."

She is also learning how to swallow again.

"Every day is a new beginning for me. I'm working so hard to use my vocal cords and train my muscles to swallow. "I'll probably never sing in a choir or anything, but it's exciting to talk normally and I cant wait to eat and drink and swim again."

What will her voice sound like?

Her new voice should sound very similar to her old one. How people talk is a combination of the way the lips and tongue move as well as how the lungs and brain function. Professor Martin Birchall, from UCL, said: "The larynx is a sound generator, acting like a reed in the clarinet. All of the other structures are the same, the only thing that's changed is the reed."

Professor Martin Birchall, from University College London, who was part of the surgery team, said: "The larynx is one of the most sophisticated neuromuscular organs in the body."

"We've learned that we can repair nerves to make even very complex organs function again. It'll open the door to better facial transplants and will be extremely important as tissue engineering develops."

A voicebox transplant might be life changing, but it is not lifesaving.

The procedure is rare, the only other documented case took place at the Cleveland Clinic in 1998.

Everyone who receives a transplant must take drugs to suppress the immune system for the rest of their lives.

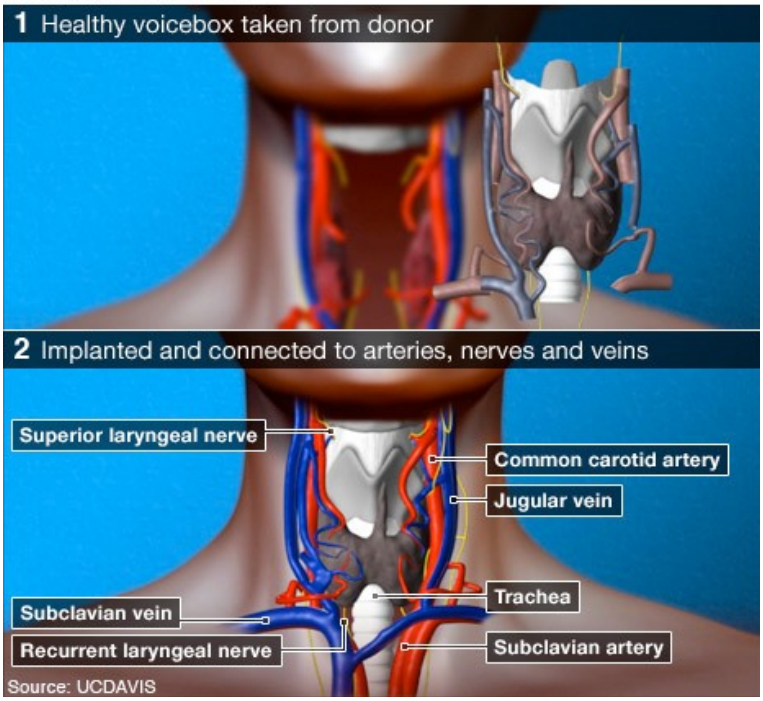
These drugs can reduce life expectancy so are normally reserved for life saving procedures.

One of the reasons Ms Jensen was suitable for this transplant was because she was already taking immunosuppresing drugs after a kidney-pancreas transplant four years ago.

Professor Peter Belafsky, part of the surgical team, said: "Brenda was an exceptional candidate for the transplant because she was highly motivated.

"Anyone who's met Brenda knows that she is a strong and determined individual with a terrific outlook on life despite the many physical challenges she's faced over her lifetime."

Voicebox transplant



http://www.physorg.com/news/2011-01-red-blood-cell-hormone-modulates.html

Red blood cell hormone modulates the immune system

New research reveals that a hormone best known for stimulating the production of red blood cells can modulate the immune response.

The study, published by Cell Press in the January 27th issue of the journal Immunity, finds that erythropoietin (EPO) has contrasting influences on infectious and inflammatory diseases and may be useful in the design of new therapeutic strategies.

EPO is a cytokine hormone that stimulates the production of red blood cells by acting at EPO receptors (EPORs) on red blood cell precursors. Interestingly, other cell types also express EPORs. "It is clear that EPORs are present on immune cells, but the function of these receptors was completely unknown," says senior study author Dr. Guenter Weiss from Innsbruck Medical University in Austria. "We hypothesized that EPO might be able to modulate the immune system and could be of clinical relevance in certain diseases."

After showing that EPO inhibited induction of key pro-inflammatory genes, Dr. Weiss and colleagues examined the role of EPO-modulated immune cells in two mouse models of disease: systemic infection with Salmonella bacteria and chemically induced inflammation of the colon (colitis).

In mice infected with Salmonella, EPO treatment was associated with reduced survival and impaired ability to clear the pathogen, neutralization of EPO production in the body promoted Salmonella elimination. This suggests that EPO reduces the ability of the immune system to fight off a systemic infection with intracellular bacteria such as Salmonella.

The researchers went on to show that in contrast to bacterial infection, EPO had a beneficial effect on the severity of colitis. EPO decreased the production of nuclear factor (NF)-□B, a protein that is critical for inflammation and thereby reduced the formation of cytokines such as tumor necrosis factor alpha which are centrally involved in the pathogenesis of autoimmune colitis. This suggests that EPO may exert beneficial effects in non-infectious inflammatory diseases.

"Our results provide novel evidence that EPO acts as a potent anti-inflammatory immune modulator by specifically targeting (NF)-□B-driven inflammatory pathways," concludes Manfred Nairz, first author of the paper. "Although high dose EPO treatment in humans may lead to a dangerous excess of red blood cells, EPO derivatives that do not influence red blood cell production have been developed and these could possibly serve as valuable therapeutic tools in treatment of pathologic inflammation." *Provided by Cell Press*

http://www.physorg.com/news/2011-01-feds-post-vaccine-seizures-young-kids.html

Feds checking post-vaccine seizures in young kids

January 20, 2011 By MIKE STOBBE , AP Medical Writer

(AP) -- ***Government officials are investigating an apparent increase in fever-related seizures in young children after they got a flu shot.***

The U.S. Food and Drug Administration on Thursday said there have been 36 confirmed reports of seizures this flu season in children ages 6 months through 2 years. The seizures occurred within one day after they were vaccinated with Fluzone, the only flu shot recommended in the United States for infants and very young children. Ten of the children were hospitalized, but all recovered.

The FDA said it is investigating to see if there is any connection between the vaccine and the seizures, or if something else caused the convulsions. The agency said recommendations for using the vaccine have not changed, nor has there been any change in flu vaccine guidance.

In the U.S., vaccination is recommended for everyone except infants under 6 months.

The vaccine's manufacturer, Sanofi Pasteur, issued a statement emphasizing that no clear link has been established between the flu shot and the seizures and that the cases may be coincidental.

Thursday's announcement comes at a time when the FDA has been working on disclosing more information about potential safety problems with drugs and devices after they've been approved.

The government uses a national reporting system to monitor possible side effects following vaccination. Doctors, nurses, parents and vaccine manufacturers all can file reports.

"It's meant to cast a wide net" to look for problems, but is only regarded as preliminary information that must be checked out, said Shelly Burgess, an FDA spokeswoman.

FDA officials said they've been paying special attention to seizure reports because of an unexplained higher rate of fevers and seizures in young children in Australia and New Zealand who got a specific flu vaccine earlier this year. In August, a U.S. vaccines advisory panel said doctors should avoid using that vaccine, made by CSL Biotherapies, in children ages 6 months through 8 years.

It's possible the Australia cases sparked increased reports this fall, said Dr. Andrea Sutherland, an official in the FDA's Center for Biologics Evaluation and Research.

The fever-related seizures - called febrile seizures - are convulsions brought on by a fever in infants or small children. A child often loses consciousness and shakes. Most seizures last a minute or two, and often children quickly recover. Such seizures may occur with any common childhood illnesses that may cause fever, such as ear infections, colds, influenza and other viral infections.

More information: FDA notice: http://tinyurl.com/FDAnotice

http://www.eurekalert.org/pub_releases/2011-01/uos-rrf012111.php

Researchers reveal function of novel molecule that underlies human deafness

New research from the University of Sheffield has revealed that the molecular mechanism underlying deafness is caused by a mutation of a specific microRNA called miR-96.

The discovery could provide the basis for treating progressive hearing loss and deafness.

The research team, led by Dr Walter Marcotti, Royal Society University Research Fellow from the University's Department of Biomedical Science, in collaboration with Professor Karen Steel at the Sanger Institute in Cambridge, discovered that the mutation in miR-96 prevents development of the auditory sensory hair cells. These cells are located in the inner ear and are essential for encoding sound as electrical signals that are then sent to the brain.

The research has been published this week in the Proceedings of the National Academy of Sciences journal and was based on studies of mice, which do not normally hear until about 12 days after birth. Prior to this age their immature hair cells must execute a precise genetic program that regulates the development of distinct types of sensory hair cell, namely inner and outer hair cells.

The research teams found that in a strain of mice called diminuendo - which carry a single base mutation in the miR-96 gene - hair cell development is arrested around birth.

The study shows that miR-96 normally regulates hair cell development by influencing the expression of many different genes associated with a wide range of developmental processes at a specific stage. The researchers discovered that the mutation hinders the development not only of the mechanically sensitive hair bundle on the cell apex but also the synaptic structures at the base that govern transfer of electrical information to the sensory nerves. These new findings suggest that miR-96 is a master regulator responsible for coordinating the development of the sensory cells that are vital to hearing.

Since the mutation in miR-96 is known to cause human deafness and microRNA molecules can be targeted by drugs, the work also raises new opportunities for developing treatments to treat hearing loss.

Dr Walter Marcotti said: "Progressive hearing loss affects a large proportion of the human population, including new born and young children. Despite the relevance of this problem, very little is currently known regarding the genetic basis of progressive hearing loss. Our research has provided new and exciting results that further our understanding of auditory development as well as possible molecular targets for the development of future therapies."

Notes for editors:

The work was supported by the Royal National Institute for Deaf People (RNID), The Wellcome Trust and the University of Sheffield.

To read the research paper, entitled miR-96 regulates the progression of differentiation in mammalian cochlear inner and outer hair cells, visit: http://www.pnas.org/content/early/2011/01/13/1016646108.abstract

For further information please contact: Shemina Davis, Media Relations Officer, on 0114 2225339 or email shemina.davis@sheffield.ac.uk

To view this news release and images online, visit http://www.shef.ac.uk/mediacentre/2011/1831-gene-deafness-mutation

http://www.nytimes.com/2011/01/21/science/21memory.html

To Really Learn, Quit Studying and Take a Test

By PAM BELLUCK

Taking a test is not just a passive mechanism for assessing how much people know, according to new research.

It actually helps people learn, and it works better than a number of other studying techniques.

The research, published online Thursday in the journal Science, found that students who read a passage, then took a test asking them to recall what they had read, retained about 50 percent more of the information a week later than students who used two other methods.

One of those methods — repeatedly studying the material — is familiar to legions of students who cram before exams. The other — having students draw detailed diagrams documenting what they are learning — is prized by many teachers because it forces students to make connections among facts.

These other methods not only are popular, the researchers reported; they also seem to give students the illusion that they know material better than they do.

In the experiments, the students were asked to predict how much they would remember a week after using one of the methods to learn the material. Those who took the test after reading the passage predicted they would remember less than the other students predicted — but the results were just the opposite.

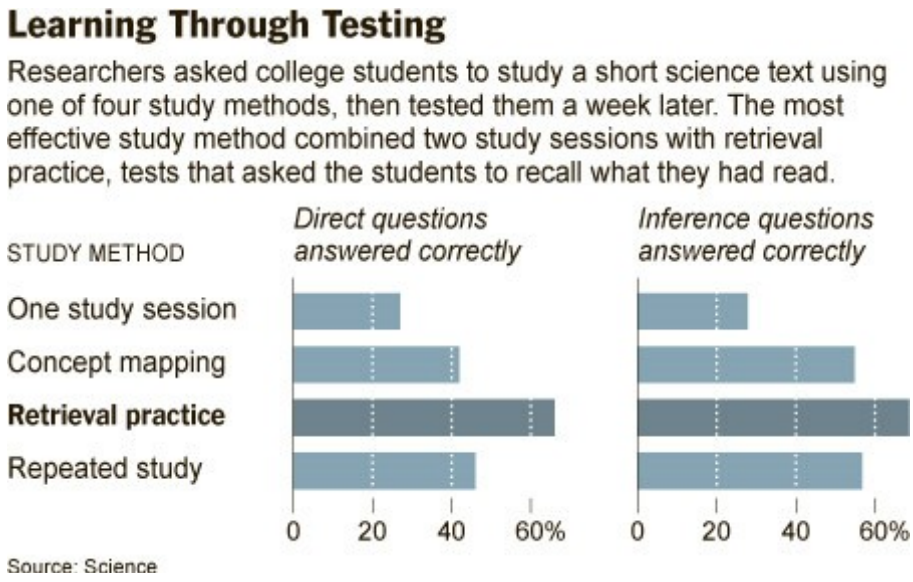
"I think that learning is all about retrieving, all about reconstructing our knowledge," said the lead author, Jeffrey Karpicke, an assistant professor of psychology at Purdue University. "I think that we're tapping into something fundamental about how the mind works when we talk about retrieval."

Several cognitive scientists and education experts said the results were striking.

The students who took the recall tests may "recognize some gaps in their knowledge," said Marcia Linn, an education professor at the University of California, Berkeley, "and they might revisit the ideas in the back of their mind or the front of their mind."

When they are later asked what they have learned, she went on, they can more easily "retrieve it and organize the knowledge that they have in a way that makes sense to them."

The researchers engaged 200 college students in two experiments, assigning them to read several paragraphs about a scientific subject — how the digestive system works, for example, or the different types of vertebrate muscle tissue.



In the first experiment, the students were divided into four groups. One did nothing more than read the text for five minutes. Another studied the passage in four consecutive five-minute sessions.

A third group engaged in "concept mapping," in which, with the passage in front of them, they arranged information from the passage into a kind of diagram, writing details and ideas in hand-drawn bubbles and linking the bubbles in an organized way.

The final group took a "retrieval practice" test. Without the passage in front of them, they wrote what they remembered in a free-form essay for 10 minutes. Then they reread the passage and took another retrieval practice test.

A week later all four groups were given a short-answer test that assessed their ability to recall facts and draw logical conclusions based on the facts.

The second experiment focused only on concept mapping and retrieval practice testing, with each student doing an exercise using each method. In this initial phase, researchers reported, students who made diagrams while consulting the passage included more detail than students asked to recall what they had just read in an essay.

But when they were evaluated a week later, the students in the testing group did much better than the concept mappers. They even did better when they were evaluated not with a short-answer test but with a test requiring them to draw a concept map from memory.

Why retrieval testing helps is still unknown. Perhaps it is because by remembering information we are organizing it and creating cues and connections that our brains later recognize.

"When you're retrieving something out of a computer's memory, you don't change anything — it's simple playback," said Robert Bjork, a psychologist at the University of California, Los Angeles, who was not involved with the study.

But "when we use our memories by retrieving things, we change our access" to that information, Dr. Bjork said. "What we recall becomes more recallable in the future. In a sense you are practicing what you are going to need to do later." It may also be that the struggle involved in recalling something helps reinforce it in our brains.

Maybe that is also why students who took retrieval practice tests were less confident about how they would perform a week later.

"The struggle helps you learn, but it makes you feel like you're not learning," said Nate Kornell, a psychologist at Williams College. "You feel like: 'I don't know it that well. This is hard and I'm having trouble coming up with this information.'" By contrast, he said, when rereading texts and possibly even drawing diagrams, "you say: 'Oh, this is easier. I read this already.'"

The Purdue study supports findings of a recent spate of research showing learning benefits from testing, including benefits when students get questions wrong. But by comparing testing with other methods, the study goes further.

"It really bumps it up a level of importance by contrasting it with concept mapping, which many educators think of as sort of the gold standard," said Daniel Willingham, a psychology professor at the University of Virginia. Although "it's not totally obvious that this is shovel-ready — put it in the classroom and it's good to go — for educators this ought to be a big deal."

Howard Gardner, an education professor at Harvard who advocates constructivism — the idea that children should discover their own approach to learning, emphasizing reasoning over memorization — said in an e-mail that the results "throw down the gauntlet to those progressive educators, myself included."

"Educators who embrace seemingly more active approaches, like concept mapping," he continued, "are challenged to devise outcome measures that can demonstrate the superiority of such constructivist approaches."

Testing, of course, is a highly charged issue in education, drawing criticism that too much promotes rote learning, swallows valuable time for learning new things and causes excessive student anxiety.

"More testing isn't necessarily better," said Dr. Linn, who said her work with California school districts had found that asking students to explain what they did in a science experiment rather than having them simply conduct the hands-on experiment — a version of retrieval practice testing — was beneficial. "Some tests are just not learning opportunities. We need a different kind of testing than we currently have."

Dr. Kornell said that "even though in the short term it may seem like a waste of time," retrieval practice appears to "make things stick in a way that may not be used in the classroom.

"It's going to last for the rest of their schooling, and potentially for the rest of their lives."

http://www.physorg.com/news/2011-01-world-internet.html

World 'running out of Internet addresses'

Vint Cerf said the world will run out of Internet addresses "within weeks"

The world will run out of Internet addresses "within weeks", according to one of the founding fathers of the web, a report said Friday.

Vint Cerf, who helped create the web by connecting computers using Internet Protocol (IP) addresses, said it was his "fault" that the 4.3 billion addresses created were running out, the Sydney Morning Herald reported.

"I thought it was an experiment and I thought that 4.3 billion would be enough to do an experiment," Cerf, who is Google's vice president and "Chief Internet Evangelist", was quoted as saying in an interview.

"Who the hell knew how much address space we needed?"

In 1977, Cerf created the web protocol IPv4, which connects computers globally, as part of an experiment while working with the US Department of Defense. He said he never expected his experiment "wouldn't end".

"It doesn't mean the network stops, it just means you can't build it very well," Cerf said.

IP addresses are the unique sequence of numbers assigned to each computer, website or other internet-connected devices. They are not the same as website domain names.

The overwhelming number of devices now accessing the internet means the addresses are running out fast.

To resolve the crisis, an updated protocol for the Internet, IPv6, currently being planned by the industry, will create trillions of addresses.

As Google vice president Cerf, who was in Australia to address a conference, said he thought the new chief executive of the California-based giant, Larry Page, was ready to lead the company into the future.

In a surprise move, Google announced on Thursday that co-founder Page would replace Eric Schmidt as chief executive in April.

Schmidt, 55, a former chief executive of Novell, will remain with Google as executive chairman, focusing on deals, partnerships, customers and government outreach, Google said.

He will also act as an adviser to Page, 37, who served as CEO previously, from 1998 to 2001.

Cerf said Schmidt had been chief executive for 10 years -- "a nice round number" -- and Page was ready to lead the company into the future.

"Larry and Sergey are 10 years older than they were when they thoughtfully hired Eric to be the CEO... so everybody's growing up," Cerf said.

Google has grown over the past decade from a start-up battling other Internet search engines into a technology giant with nearly 25,000 employees and annual revenue of nearly $30 billion.

The company meanwhile reported its fourth-quarter net profit increased to $2.54 billion from $1.97 billion a year ago, while revenue rose 26 percent to $8.44 billion.

http://www.nytimes.com/2011/01/20/health/research/20cancer.html

Drug to Fight Melanoma Prolonged Life in Trial

By AMY HARMON

Advanced melanoma patients taking an experimental drug aimed at a particular mutation in their tumors lived longer than patients who did not receive the drug in a decisive clinical trial, the drug's manufacturer, Roche, said Wednesday.

The results pave the way for Roche to seek approval to market the drug, which shrank tumors for an average of six months in earlier trials but had not yet been proven to prolong survival. Developed by Plexxikon, a small biotechnology company in Berkeley, Calif., it is based on an understanding of cancer's most basic molecular workings that is seen as a potential key to providing more lasting treatments for melanoma and other cancers.

"In the past, with chemotherapy, we were grasping at things," said Dr. Paul Chapman, an oncologist at Memorial Sloan-Kettering Cancer Center who led the trial. "Now we have a rational way of building on this. For the first time, we can see the path forward."

About half of the 68,000 Americans who develop melanoma every year have a mutation in a gene, called B-RAF, that goes awry, for reasons not well understood, signaling cells to grow uncontrollably. The Roche drug works by blocking a malfunctioning protein the gene produces in cancer cells, but leaving the functioning proteins in noncancerous cells alone.

In the drug's earliest trial, nearly every patient whose tumor cells contained the B-RAF mutation responded to the drug. That marked a radical difference from standard chemotherapies, whose reason for working in certain patients and not others is not well understood.

It also led some oncologists to assail the ethics of the trial, whose early results were disclosed on Wednesday, because it required some patients who might have been helped by the new drug to instead take a chemotherapy drug that was seen as essentially a placebo. Starting in January of last year, 338 patients were assigned to the chemotherapy arm of the trial, while another 338 received the Roche drug. A series of articles in The New York Times last year described the early testing of the drug and the debate among oncologists over the design of the latest trial.

To measure whether the drug prolonged lives, the company was to compare survival on both arms of the trial. A Food and Drug Administration spokeswoman said Wednesday that the agency asked the company to perform that analysis sooner than originally planned. Patients on the chemotherapy arm of the trial will now be able to "cross over" and receive the drug.

Melanoma, the deadliest form of skin cancer, has been essentially untreatable after it spreads, with a median survival rate of eight months from the time of diagnosis. Roche has not yet reported how much longer patients taking its drug live, on average. The company has opened an "expanded access" program at three cancer centers to provide the drug for melanoma patients who are not enrolled on the trial.

But even as patient advocates and oncologists welcomed the results of the Roche trial on Wednesday, they called for more expedient testing of drugs to combine with it. Research into why patients ultimately relapse after responding to the drug is pointing to other drugs similarly tailored to particular mutations that could yield more durable benefits for patients. "Combining drugs is where the future of oncology treatment lies," said Timothy Turnham, executive director of the Melanoma Research Foundation, a nonprofit advocacy group. "We need companies to cooperate and make this happen now."

http://www.physorg.com/news/2011-01-clinical-trials-cited-previous-relevant.html

Clinical trials cited for ignoring previous relevant research

The vast majority of already published and relevant clinical trials of a given drug, device or procedure are routinely ignored by scientists conducting new research on the same topic, a new Johns Hopkins study suggests.

The vast majority of already published and relevant clinical trials of a given drug, device or procedure are routinely ignored by scientists conducting new research on the same topic, a new Johns Hopkins study suggests.

The authors of the findings, reported in the Jan. 4 issue of Annals of Internal Medicine, argue that these omissions potentially skew scientific results, waste taxpayer money on redundant studies and involve patients in unnecessary research.

Conducting an analysis of published studies, the Johns Hopkins team concludes that researchers, on average, cited less than 21 percent of previously published, relevant studies in their papers. For papers with at least five prior publications available for citation, one-quarter cited only one previous trial, while another quarter cited no other previous trials on the topic. Those statistics stayed roughly the same even as the number of papers available for citation increased. Larger studies were no more likely to be cited than smaller ones.

"The extent of the discrepancy between the existing evidence and what was cited is pretty large and pretty striking," says Karen Robinson, Ph.D., an assistant professor of medicine at the Johns Hopkins University School of Medicine and co-author of the research with Steven N. Goodman, M.D., M.H.S., Ph.D., a Hopkins epidemiology and biostatistics professor. "It's like listening to one witness as opposed to the other 12 witnesses in a criminal trial and making a decision without all the evidence. Clinical trials should not be started — and cannot be interpreted — without a full accounting of the existing evidence."

Robinson and Goodman searched the Web of Science, an Internet archive, for meta-analyses done in 2004 on groups of randomized, controlled trials on such common topics as a cancer treatment or a heart procedure. A meta-analysis is a systematic procedure for statistically combining the results of many different studies on a similar topic to determine the effectiveness of medical interventions.

The researchers ultimately looked at 227 meta-analyses comprising 1,523 separate clinical trials in 19 different fields, including oncology, neurology and pediatrics. Of 1,101 peer-reviewed publications for which there had been at least five previous relevant papers, 46 percent acknowledged the existence of no more than one previous trial.

"Accurate representation of the prior cumulative evidence is necessary to both ethically justify a trial and to make proper inferences," they write. Studying prior research can also lead to study designs that are more likely to fill gaps in the evidence, the team said, noting that although the presence of a citation "does not tell us how information from that trial was used, the absence of a citation almost guarantees that it was not."

The Hopkins researchers could not say why prior trials failed to be cited or whether non-cited trials may have been taken into account in the trial design and proposal stages, such as grant requests to the National Institutes of Health.

At the very least, Robinson says, researchers often contend that their publications are so "unique" that there are no relevant studies to cite, even though someone else may have included it in a meta-analysis of like research. Others claim there just isn't room to cite past relevant research, but Robinson says one reason for the omissions could be the self-interest of researchers trying to get ahead.

"To get published, journals are looking for novelty, uniqueness," she says. Leaving out references to prior similar research can make findings seem more like a breakthrough, she adds. In her publications study, Robinson found several papers that claimed to be the first even when many trials on the subject preceded them. There are no barriers to funding, conducting or publishing a clinical trial without proof that prior literature had been adequately searched and evaluated, she says. But requirements such as those have been instituted by some European funding agencies, the medical journal The Lancet, and the U.S. Centers for Medicare & Medicaid Services, which require that a covered trial not "unjustifiably duplicate existing studies," Robinson writes.

Robinson says funders, institutional review boards and journals need to take steps to ensure that prior research is considered. To do otherwise, she says, encourages this "unethical" behavior to continue.

"Trials being done may not be justified, because researchers are not looking at or at least not reporting what is already known," she says. "We may be wasting resources when we fund trials for which we already know the answer. And we may be coming to incorrect conclusions about what works in medicine."

In some cases, patients who volunteer for clinical trials may be getting a placebo for a medication that a previous researcher has already determined works or may be getting a treatment that another researcher has shown is of no value. In rare instances, patients have suffered severe side effects and even died in studies because researchers were not aware of previous studies documenting a treatment's dangers.

Provided by Johns Hopkins University

http://www.physorg.com/news/2011-01-gold-eye-solar-energy.html

Fool's gold catches eye of solar energy researchers

Iron pyrite - also known as fool's gold - may be worthless to treasure hunters, but it could become a bonanza to the solar industry.

The mineral, among the most abundant in the earth's crust, is usually discarded by coal miners or sold as nuggets in novelty stores. But researchers at the University of California-Irvine said they could soon turn fool's gold into a cheaper alternative to the rare and expensive materials now used in making solar panels.

"With alternative energy and climate-change issues, we're always in a race against time," said lead researcher Matt Law. "With some insight and a little bit of luck, we could find a good solution with something that's now disposed of as useless garbage."

The UC-Irvine team believes the mineral can be processed into a thin film for use in photovoltaic cells, and could eventually convert sunlight into electricity at roughly the same rate as existing technology.

Though it's too early to estimate the cost of cells made with pyrite, Law said they're likely to be cheaper because fool's gold is so readily available. A prototype could be ready within the year, but it could be at least three years before the cells are commercially available.

Some industry analysts, however, are skeptical that the team - which includes a chemist, a mathematician and a physicist - can hit pay dirt.

"I don't want to pour cold water on what they're doing, but every day somebody comes up with a new idea for a solar cell technology," said Shyam Mehta, a solar industry analyst with GTM Research. "Commercializing it is a lot more difficult than people seem to think, and it's full of failed attempts."

To be successful in the market, he said, scientists have to replicate the carefully controlled conditions of a laboratory in a factory capable of producing hundreds of thousands of panels a year, at a cost that can compete with Chinese prices.

The U.S. solar photovoltaics industry is worth at least $2 billion and growing, but not much of the cell-making process occurs domestically. Existing types of cells, such as cadmium telluride and amorphous silicon, use materials that are either very scarce, potentially toxic or not especially efficient.

And other materials such as indium - about $300 a pound - are in high demand for use in touch screens and other tech gadgets. These so-called rare earth elements are available only from a single U.S. mine in California or from China, which is clamping down on exports of the material.

Law and his colleagues believe fool's gold, which is composed of iron and sulfur, could be used to make solar cells in a major production process.

Iron pyrite has been eyed as a candidate for solar panels in scattered studies in the 1980s and '90s, along with other cheaper, abundant materials such as copper oxide, copper sulfide and zinc phosphide, Law said. But a lack of clean-tech financing, unsophisticated processing equipment and lack of interest caused the research efforts to fizzle.

"Now, with better tools and funding and a sense of urgency, more people are looking again at very promising materials that might have had one stumbling block or two earlier that had tripped them up," Law said.

One of the challenges in developing solar cells from fool's gold is that the material has poor voltage. That is, the mineral is full of microscopic pockets that suck in electrons, limiting conductivity and the ability to convert solar energy into electricity. Law's team is working on ways to plug the holes.

The work is being funded in part by a three-year grant from the National Science Foundation's solar program.

Law said the effort is attracting the attention of solar companies and other researchers, many of whom are starting to look into iron pyrite again. But with existing photovoltaic technology already so established, new solar innovations will have a harder time catching up in the market, he said.

"There's a narrowing window for new technology to come online," Law said. "If we fall asleep at the switch, it'll be much more difficult to compete against big companies that are already learning to do this better, more efficiently and faster."

http://www.physorg.com/news/2011-01-teeth-stem-cells.html

Pulled teeth stored for stem cells

Naidelys Montoya didn't wait for her son's baby teeth to fall out. She took the boy to an oral surgeon to have two of the loose ones extracted.

"He was a bit scared," said Montoya, of Hialeah, Fla. "He's not that brave."

The dentist shipped the teeth in a temperature-controlled steel container to a lab in Massachusetts, where their stem cells will be spun out, frozen to more than 100 degrees below zero and stored - in case her son, Raul Estrada, 6, might need them for a future illness.

"I believe in this," Montoya said. "I did as a precaution against things that could happen."

Montoya and her son have joined a major new medical movement.

In South Florida and around the world, dentists are extracting baby teeth, wisdom teeth and even healthy adult teeth, and researchers are spinning out stem cells that they believe can be used to regrow lost teeth, someday even to repair damaged bones, hearts, pancreases, muscles and brains.

It could put the Tooth Fairy out of business.

"These are teeth we've been discarding as dental waste," said Dr. Jeffrey Blum, the Miami Beach oral surgeon who pulled Raul's teeth. "We might as well get some use out of them."

"I can't help but feel excitement for their potential use in regenerating different tissues in the human body," said Dr. Jeremy Mao, director of the Regenerative Medicine Laboratory at Columbia University. Mao also is

chief science advisor to StemSave, a New York City company that freezes the stem cells and stores them for later use.

There are concerns. It's expensive, costing $590 upfront plus $100 a year to store the stem cells from up to four teeth for up to 20 years. It's speculative, with the first FDA-approved practical use of such stem cells years away.

"Every treatment using dental stem cells is still in the clinical testing phase, and won't be ready for general use for at least five years," said Art Greco, StemSave's CEO.

Montoya understands: "Things are evolving so quickly, who knows what they will be able to do in 15 or 20 years?"

Other researchers welcome the new source of stem cells.

"Perhaps it does make sense to save" dental stem cells, said Dr. Joshua Hare, director of the Interdisciplinary Stem Cell Institute at the University of Miami Medical School, who is not involved with dental stem cells. "Within human adults and children there are lots of reservoirs of stem cells. We get them from bone marrow; others use umbilical cord blood. It seems teeth are also a good source."

The National Institutes of Health concluded in 2003 that teeth are a rich source of stem cells. Every child has about 20 baby teeth that fall out between ages 6 and 12. Adolescents have wisdom teeth that often are removed between ages 14 and 25 because they crowd the jaw or grow in crookedly.

Blum and other oral surgeons must extract baby teeth before they fall out naturally, so they still have a blood supply to keep them healthy. He puts them in a temperature-controlled steel container and overnights them to the StemSave facility.

Stem cells are the body's repair system, Hare said. Stem cells beneath the skin are constantly spinning off new skin cells to replace skin that is sloughed off or damaged in daily life. The same is true for hearts, livers, pancreases - except that as the body weakens from age, injury or disease, those stem cells start to lose the ability to keep up and need help. Today, stem cells from bone marrow, blood and now perhaps teeth can be reprogrammed to help those ailing organs.

Also, by using these stem cells, researchers avoid involving human embryonic stem cells, which are controversial because their creation involves destroying human embryos.

The first practical use of dental stem cells probably will be to repair human teeth and jawbones, researchers say. At Boston University's School of Dental Medicine, researchers have used stem cells from baby and wisdom teeth to generate dental pulp, the soft interior of a tooth, and dentin, its hard white casing.
Now they are inserting the material into a broken human tooth and implanting it into a mouse to access a blood supply. When the technology reaches humans, the pulp material would be injected into a spongy "scaffold" where a tooth has been removed and prompted to grow into a human tooth. It's at least five years away.

Across the world, the use of stem cells to heal the human body is exploding. At the University of Miami's med school, Hare is doing human trials using stem cells from bone marrow to inject around hearts damaged by heart attacks, hoping to regenerate damaged heart tissue.

For years, stem cells from umbilical cord blood have saved the lives of patients with leukemia, lymphoma, multiple myeloma, aplastic anemia, sickle cell and other diseases.

Umbilical cord blood is being donated both to private labs for use only by the donor's family, and also to public donation centers.

In Broward County, Memorial Health Care System, Memorial Hospital West in Pembroke Pines, Memorial Regional in Hollywood and Memorial Hospital in Miramar have opened or are opening public cord-blood donation centers.

Women giving birth may donate their umbilical cords without charge. The blood is flown to a lab at Duke University in North Carolina, where the stem cells are spun off and stored at subfreezing temperatures. The cells become part of a National Cord Blood bank where they are available to any patient in the world if an adequate cell match can be determined.

Cord blood stem cells collected for private use have been more speculative because of the rarity of diseases it can treat. A 2009 study published in the peer-reviewed journal Obstetrics and Gynecology said cord blood stem cells in private banks have been used in less than half of 1 percent of cases over the past 10 years. But stem cells in public cord blood cell banks are in short supply, especially for Hispanics and African Americans.

So far, only private banks are storing dental stem cells, although Mao says a public bank would be valuable and appropriate.

The American Dental Association, while cautiously optimistic about the potential of dental stem cells, urges parents considering banking their children's dental stem cells to consider both the cost and the rarity of use before joining private donation programs.

"That's the question people have to ask themselves," Blum said. "Am I saving this for no reason? Is it worth what I'm paying? Essentially it's an insurance policy." *(c) 2011, The Miami Herald.*

http://www.nytimes.com/2011/01/22/nyregion/22science.html

An Infusion of Science Where the Arts Reign

By LISA W. FODERARO

ANNANDALE-ON-HUDSON, N.Y. — *Liza Batkin, a first-year student at Bard College, is a dancer whose academic interests run to philosophy and literature. But here she was in a laboratory on a snowy day in January — while many of her peers across the country were still on winter break — inserting a piece of DNA into bacteria with a micropipette.*

At least she had company. In an intensive new program, she and every other freshman at Bard, a campus with a decidedly arty bent, have had to spend six hours a day over the past two and a half weeks learning about science through the prism of infectious disease.

The 480 students have studied under two dozen scientists recruited from across the country for the program. Using lab equipment, computer modeling and classroom discussions, they have explored all aspects of disease, including detecting germs and managing pandemics.

“There are mixed opinions, from total apathy - ‘Why am I here? This isn’t why I came to Bard’ - to total enthusiasm,” Ms. Batkin said of her classmates. “I decided to take it 100 percent seriously; otherwise I knew I wouldn’t get anything out of it. I definitely find myself becoming more critical of the science articles I read.”

Called Citizen Science, the new program is the brainchild of Bard’s president, Leon Botstein, who is himself an artist — the music director and conductor of the American Symphony Orchestra. Dr. Botstein has accused colleges of shirking their responsibility to create a well-rounded citizenry.

“The most terrifying problem in American university education is the profound lack of scientific literacy for the people we give diplomas to who are not scientists or engineers,” he said. “The hidden Achilles’ heel is that while we’ve found ways to educate scientists in the humanities, the reverse has never really happened. Everybody knows this, but nobody wants to do anything about it.”

Bard already has general-education requirements that include a semester of science, and the college has come up with some clever, though still rigorous, science courses for the nonmajor. There is one on color and light that appeals to artists, and another on acoustics for musicians.

But Dr. Botstein set out last year to do more. He consulted several scientists, including his brother, David Botstein, a renowned geneticist at Princeton University, on the notion of a brief, hands-on immersion that eventually became the Citizen Science program. “I asked, ‘Can you make a real dent in the understanding of the layperson in three weeks, intensively every day?’ ” Dr. Botstein recalled. “And to a person, they all said, ‘Yes.’ ”

Groups that promote American competitiveness have long warned that the United States is slipping in all areas of science. The National Academy of Sciences, in an update of its landmark 2005 report “Rising Above the Gathering Storm,” said last fall that the United States faced a future of economic decline if it failed to make significant investments in research in and teaching of science and mathematics.

The update, subtitled “Rapidly Approaching Category 5,” noted that 16 percent of American college students received undergraduate degrees in natural sciences or engineering in 2006, the most recent year for which data was available. That compared with 47 percent in China, 38 percent in South Korea and 27 percent in France. The original report called for the creation of 25,000 undergraduate scholarships a year in math, engineering and science. The updated report said Congress had taken some steps to implement that recommendation.

Jay B. Labov, a senior adviser for education and communication at the National Academy of Sciences and the National Research Council, said that most colleges required some science instruction, but that the course work did not always have a lab component. “Being lectured at is just fine for some students, but data shows that they retain about 10 percent of that,” Dr. Labov said. “Bard and a number of other places are recognizing that if you engage students early in their careers, you can get them hooked on science.”

Still, the Bard sessions, which ended on Friday, featured several lectures, including one from David Botstein on “the fruits of the genome sequences for society,” and an open debate on vaccinations. Future programs may focus on climate change or energy.

To promote learning for learning’s sake, students will receive neither course credits nor grades.

“This way they are not constantly worried that ‘I’m not going to do well and that it will bring down my G.P.A.,’ ” said Brooke Jude, an assistant professor of biology and the director of Citizen Science. “I think we can motivate them for something other than an A.”

Lauren Cain, 19, of Memphis, who plans to major in film and music, approached the program somewhat grudgingly. “I was pretty frustrated about losing two and a half weeks of my break,” she said. “Since I’m not a science person, I was a bit intimidated by the fact that we were going to be doing science, science, science.”

But she fell under the spell of her instructor, Stephanie B. Stockwell, a microbiologist from James Madison University, and found herself fascinated by the antimicrobial substances she handled.

While there is no final grade, there is a final project, and Ms. Batkin and six classmates came up with an idea that is pure Bard: a dance performance that illustrates how an influenza vaccine works. Students assumed the roles of the antigen, B cell, T cell and antibodies.

“We’re using rubber bands and bubbles to show the B cell alerting the T cell that there’s a foreign invader,” Ms. Batkin said. “I’m narrating the process, but I am also the antibody at the end.”

http://news.discovery.com/animals/chimp-face-personality-110122.html

Chimps Wear Personalities on their Mugs

People can gauge aggressiveness in their evolutionary cousins' expressionless faces.

content provided by Bruce Bower, Science News

In chimpanzees, as in humans, faces are personality billboards, a new study suggests.

People can usually tell whether or not a chimp acts dominantly and is physically active simply by looking at a picture of the ape's expressionless mug, says a research team led by psychologist Robert Ward of Bangor University, Wales.

Consistent with earlier evidence from other researchers, Ward and his colleagues reported last year that volunteers can also accurately detect whether people are extroverted, emotionally stable, agreeable and imaginative by looking at pictures of their neutral-looking faces. Extroversion in people and dominance in chimps both relate to assertiveness and sociability, and both partly derive from an individual's genetic makeup.

An ability to discern key personality traits via facial structure evolved more than 7 million years ago in a shared ancestor of people and chimps, the researchers propose in a paper published online Jan. 14 in Evolution and Human Behavior.

"The fact that chimpanzee facial signals can be read by humans suggests that our ability to read others' faces accurately is not solely acquired through culture, but is part of an evolved system," Ward says.

That's an intriguing hypothesis in need of testing with composite images that digitally combine many pictures of the same chimps into single mug shots, remarks psychologist and chimp researcher Lisa Parr of Emory University in Atlanta. Composites minimize slight variations from one photograph to another in lighting, skin hue, head angles and other factors that can create different personality impressions of the same individual, Parr says.

Ward and his colleagues had participants evaluate composite images of people, but technical difficulties stymied their attempts to create composite chimp faces. Anatomical landmarks used to create composite images, such as the jaw's precise position, are difficult to measure on chimps' hairy faces, Ward says. Also, composites smooth out facial textures, so chimps' faces look blurry rather than hairy, he notes.

In the new study, Ward's group conducted four experiments with a total of 139 college students. Volunteers viewed pairs of mug shots showing chimps previously identified in behavioral observations as high or low in dominance. Each photographed chimp looked straight ahead or at a slight angle, with no teeth showing and no strong shadowing over the eyes that might impart a menacing look.

Participants distinguished dominant from nondominant chimps more often than would have been expected by chance. Average accuracy rates ranged from about 60 percent to 70 percent, with higher scores for faces of male chimps than for female chimps.

The students accurately distinguished mug shots of extroverted women from those of introverted women about three-quarters of the time. Pairs of pictures came from women who reported many similar personality traits on a questionnaire, except for contrasting levels of extroversion.

Core facial characteristics of dominant chimps and extroverted people remain poorly understood, Ward says. A well-defined jawline and other outer features of the face aid in detecting extroverted women, but volunteers still do pretty well at distinguishing outgoing from shy females when shown only each person's eyes, nose and mouth.

Ward suspects that chimps who look at other chimps' expressionless faces can tell which ones act dominantly. His team plans to explore this possibility.

http://www.eurekalert.org/pub_releases/2011-01/uorm-ufo012111.php

Unexpected find opens up new front in effort to stop HIV

HIV's trickery within the macrophage revealed

HIV adapts in a surprising way to survive and thrive in its hiding spot within the human immune system, scientists have learned. While the finding helps explain why HIV remains such a formidable foe after three decades of research – more than 30 million people worldwide are infected with HIV – it also offers scientists a new, unexpected way to try to stop the virus.

The work by researchers at the University of Rochester Medical Center and Emory University was published Dec. 10 in the Journal of Biological Chemistry.

It's thanks largely to its ability to hide out in the body that HIV is able to survive for decades and ultimately win out against the body's relentless immune assault. One of the virus's favorite hiding spots is an immune cell called a macrophage, whose job is to chew up and destroy foreign invaders and cellular debris.

For more than 15 years, Baek Kim, Ph.D., has been fascinated by HIV's ability to take cover in a cell whose very job is to kill foreign cells. In the last couple of years Kim, professor of Microbiology and Immunology at the University of Rochester Medical Center, has teamed with Emory scientist Raymond F. Schinazi, Ph.D., D.Sc., director of the Laboratory of Biochemical Pharmacology at Emory's Center for AIDS Research, to test whether the virus is somehow able to sidestep its usual way of replicating when it's in the macrophage.

The pair found that when HIV faces a shortage of the molecular machinery needed to copy itself within the macrophage, the virus adapts by bypassing one of the molecules it usually uses and instead tapping another molecule that is available.

Normally, the virus uses dNTP (deoxynucleoside triphosphate, the building blocks for making the viral genetic machinery) to get the job done, but dNTP is hardly present in macrophages – macrophages don't need it, since they don't replicate. But macrophages do have high levels of a closely related molecule called rNTP (ribonucleoside triphosphate), which is more versatile and is used in cells in a variety of ways. The team found that HIV uses primarily rNTP instead of dNTP to replicate inside macrophages.

"The virus would normally just use dNTP, but it's simply not available in great quantities in the macrophage. So HIV begins to use rNTP, which is quite similar from a chemical perspective. This is a surprise," said Kim. "The virus just wants to finish replicating, and it will utilize any resource it can to do so."

When the team blocked the ability of the virus to interact with rNTP, HIV's ability to replicate in macrophages was slashed by more than 90 percent.

The work opens up a new front in the battle against HIV. Current drugs generally target dNTP, not rNTP, and take aim at the infection in immune cells known at CD4+ T cells. The new research opens up the possibility of targeting the virus in macrophages – where the virus is out of reach of most of today's drugs.

"The first cells that HIV infects in the genital tract are non-dividing target cell types such as macrophages and resting T cells" said Kim. "Current drugs were developed to be effective only when the infection has already moved beyond these cells. Perhaps we can use this information to help create a microbicide to stop the virus or limit its activity much earlier."

Kim notes that a compound that targets rNTP already exists. Cordycepin in an experimental compound, derived from wild mushrooms, that is currently being tested as an anti-cancer drug. The team plans to test similar compounds for anti-HIV activity.

"This significant breakthrough was unappreciated prior to our paper. We are now exploiting new anti-HIV drugs jointly based on this novel approach that are essentially not toxic and that can be used to treat and prevent HIV infections," said Schinazi, who has developed several of the drugs currently used to treat HIV patients.

The first authors of the paper, who contributed equally to the project, are graduate students Edward Kennedy of Rochester and Christina Gavegnano of Emory. Other authors include, from Rochester, graduate students Laura Nguyen, Rebecca Slater and Amanda Lucas; and from Emory, post-doctoral associate Emilie Fromentin.

The work was funded by the National Institute of Allergy and Infectious Diseases and the U.S. Department of Veterans Affairs, where Schinazi is also employed.

http://www.eurekalert.org/pub_releases/2011-01/uoc-rfs012111.php

Researchers find smoking gun of world's biggest extinction

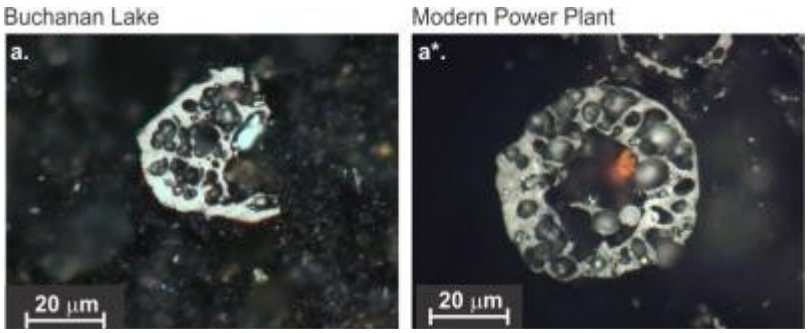
Massive volcanic eruption, burning coal and accelerated greenhouse gas choked out life

About 250 million years about 95 per cent of life was wiped out in the sea and 70 per cent on land. Researchers at the University of Calgary believe they have discovered evidence to support massive volcanic eruptions burnt significant volumes of coal, producing ash clouds that had broad impact on global oceans.

"This could literally be the smoking gun that explains the latest Permian extinction," says Dr. Steve Grasby, adjunct professor in the University of Calgary's Department of Geoscience and research scientist at Natural Resources Canada.

Grasby and colleagues discovered layers of coal ash in rocks from the extinction boundary in Canada's High Arctic that give the first direct proof to support this and have published their findings in Nature Geoscience.

The coal ash particle on the left is from the latest Permian extinction boundary at Buchanan Lake. Nunavut, the particle on the right is from a modern power plant. **Credit: Hamed Sanei, NRCan/University of Calgary**



Unlike end of dinosaurs, 65 million years ago, where there is widespread belief that the impact of a meteorite was at least the partial cause, it is unclear what caused the late Permian extinction. Previous researchers have suggested massive volcanic eruptions through coal beds in Siberia would generate significant greenhouse gases causing run away global warming.

"Our research is the first to show direct evidence that massive volcanic eruptions – the largest the world has ever witnessed –caused massive coal combustion thus supporting models for significant generation of greenhouse gases at this time," says Grasby.

At the time of the extinction, the Earth contained one big land mass, a supercontinent known as Pangaea. The environment ranged from desert to lush forest. Four-limbed vertebrates were becoming more diverse and among them were primitive amphibians, early reptiles and synapsids: the group that would, one day, include mammals.

The location of volcanoes, known as the Siberian Traps, are now found in northern Russia, centred around the Siberian city Tura and also encompass Yakutsk, Noril'sk and Irkutsk. They cover an area just under two-million-square kilometers, a size greater than that of Europe. The ash plumes from the volcanoes traveled to regions now in Canada's arctic where coal-ash layers where found.

Grasby studied the formations with Dr. Benoit Beauchamp, a professor in the Department of Geoscience at the University of Calgary. They called upon Dr. Hamed Sanei adjunct professor at the University of Calgary and a researcher at NRCan to look at some of peculiar organic layers they had discovered.

"We saw layers with abundant organic matter and Hamed immediately determined that they were layers of coal-ash, exactly like that produced by modern coal burning power plants," says Beauchamp.

Sanei adds: "Our discovery provides the first direct confirmation for coal ash during this extinction as it may not have been recognized before."

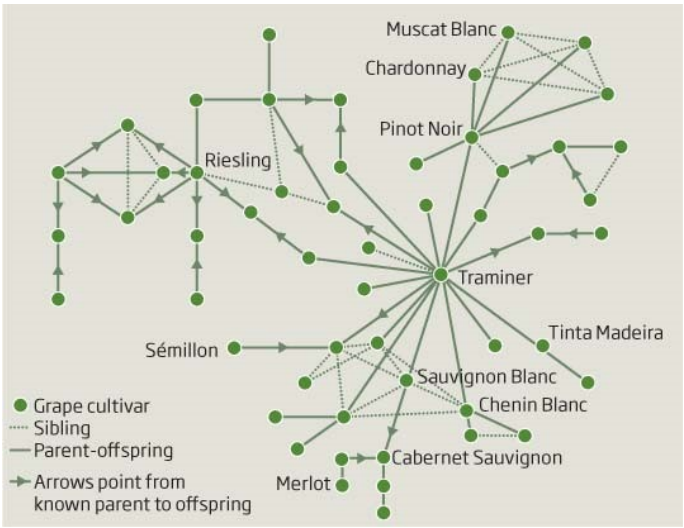
The ash, the authors suggest, may have caused even more trouble for a planet that was already heating up with its oceans starting to suffocate because of decreasing oxygen levels.

"It was a really bad time on Earth. In addition to these volcanoes causing fires through coal, the ash it spewed was highly toxic and was released in the land and water, potentially contributing to the worst extinction event in earth history," says Grasby.

http://www.newscientist.com/article/mg20927964.000-wine-family-tree-revealed.html

Wine family tree revealed

FROM Riesling to Merlot, wine grapes from around the world are more closely related than expected, says the largest study so far to produce a family tree of grapes.



The tree, above, also reveals that in 6000 years of domestication, breeders have left a vast swathe of possible varieties unexplored.

Sean Myles of Stanford University in California and colleagues looked at 9000 genetic markers in each of the world's 583 cultivated grape varieties, or cultivars, to draw up the tree.

They found that unlike other domesticated crops, most of the main cultivars are close cousins of one another. This was true regardless of where they are grown (Proceedings of the National Academy of Sciences, DOI: 10.1073/pnas.1009363108).

Moreover, breeders have been unimaginative in the crosses they have made, reusing the same cultivars over and over. The Traminer cultivar, for example, has been bred for millennia and has 20 first-degree relatives. This is good news for breeders seeking to develop cultivars that are resistant to disease, says Myles, as so few of the potential crosses have actually been made.

http://www.bbc.co.uk/news/health-12224172

TB vaccine protects before and after exposure

A new vaccine that can fight tuberculosis (TB) before and after infection has been developed by Danish scientists.

It could offer protection for many years more than is now possible.

TB is a huge global problem, particularly in developing countries, where access to antibiotics to treat the disease is limited. The latest vaccine, so far tested in animals, is featured in the journal Nature Medicine.

TB is a disease of the lungs, causing symptoms such as coughing, chest pains and weight loss. Untreated, it can be deadly. However, only in a small number of cases - fewer than 5% - do the symptoms develop immediately after infection.

In more than 90% of cases, once Mycobacterium tuberculosis, the bacterium which causes the disease, has invaded the body it changes its chemical signature, and lives in a dormant - or "latent" - state.

Usually the bacterium never emerges from this latent state, but in around 10% of cases it reactivates - often years or even decades later - to trigger severe symptoms.

Current vaccines, such as the BCG vaccine, work only if given before exposure to the bacterium.

They do not prevent infection, but do prevent acute symptoms and disease from emerging.

But once the bacterium has changed into its latent form it is effectively immune to the vaccine, and can bide its time, reactivating after the vaccine has ceased to have a preventative effect.

If successful in human trials, the new vaccine would be able to tackle that problem.

'Major breakthrough'

Developed by a team at the Statens Serum Institute in Copenhagen, it combines proteins that trigger an immune response to both the active and latent forms of Mycobacterium.

Researcher Professor Peter Lawætz Andersen said: "It might be possible to give a booster jab post-exposure to older children or even young adults which would protect them well into adulthood."

Although TB can be treated with antibiotics, those drugs are often not easily accessible in the developing world, where the new vaccine could have the greatest benefit.

Professor Andersen said: "In these areas you cannot go in and treat more than half the local population. For instance, in Capetown 60% of people are thought to be infected."

Tuberculosis
Tuberculosis is an infectious disease that usually affects the lungs
It is transmitted via droplets from the lungs of people with the active form of the disease
Symptoms of TB include coughing, chest pains, weakness, weight loss, fever and night sweats
Tuberculosis is treatable with a course of antibiotics

Professor Peter Davies, secretary of the group TB Alert, said: "A vaccine which can both protect against initial infection and protect from a breakdown of infection into disease is a major breakthrough.

"One of the main disadvantages of BCG was that it could only prevent infection going on to disease in the initially uninfected individual. It was therefore of no use in protecting infected adults who would become an infectious source of disease. Protecting children, though of value, does not protect against transmission, as children with active disease do not usually transmit disease.

"So far so good but we must remember that mice are not men (or women)."

Professor Francis Drobniewski, Director of the Health Protection Agency's National Mycobacterium Reference Laboratory said: "This is an exciting and thoughtful piece of research. The existing BCG vaccine is cheap, safe, widely used but of limited efficacy.

"With over nine million new TB cases globally each year and increasing levels of drug resistance new diagnostics, drugs and especially effective vaccines are desperately needed."

UK situation

The number of tuberculosis cases in the UK topped 9,000 in 2009 - the highest for nearly 30 years.

Diagnoses have been rising almost continuously since the 1980s, with many of the new cases thought to be among people who caught the disease abroad.

There has also been a sharp rise in drug-resistant TB cases.

The Health Protection Agency has warned more efforts must be made to curb the problem.

http://www.eurekalert.org/pub_releases/2011-01/iu-ish012111.php

IU study: Humans' critical ability to throw long distances aided by an illusion

BLOOMINGTON, Ind. -- ***Can't help molding some snow into a ball and hurling it or tossing a stone as far into a lake as you can? New research from Indiana University and the University of Wyoming shows how humans, unlike any other species on Earth, readily learn to throw long distances.***

This research also suggests that this unique evolutionary trait is entangled with language development in a way critical to our very existence.

The study, appearing online Jan. 14 in the journal "Evolution and Human Behavior," suggests that the well-established size-weight illusion, where a person who is holding two objects of equal weight will consider the larger object to be much lighter, is more than just curious or interesting, but a necessary precursor to humans' ability to learn to throw -- and to throw far.

Just as young children unknowingly experience certain perceptual auditory biases that help prepare them for language development, the researchers assert that the size-weight illusion primes children to learn to throw. It unwittingly gives them an edge -- helping them choose an object of size and weight most effective for throwing.

"These days we celebrate our unique throwing abilities on the football or baseball field or basketball court, but these abilities are a large part of what made us successful as a species," said Geoffrey Bingham, professor in IU's Department of Psychological and Brain Sciences. "It was not just language. It was language and throwing that led to the survival of Homo sapiens, and we are now beginning to gain some understanding of how these abilities are rapidly acquired by members of our species."

Why is throwing so important from an evolutionary standpoint? Bingham said Homo sapiens have been so successful as a species because of three factors: Social organization and cooperation, language, which helps with the former factor, and the ability to throw long distance. This trio allowed Homo sapiens to "take down all the potential competition," Bingham said. It brought us through the ice ages because Homo sapiens could hunt the only major food sources available, big game such as mammoths and giant sloths.

Bingham and Qin Zhu, lead author of the study and assistant professor at the University of Wyoming, Laramie, consider throwing and language in concert, because both require extremely well-coordinated timing and motor skills, which are facilitated by two uniquely developed brain structures -- the cerebellum and posterior parietal cortex.

"The idea here is that our speech and throwing capabilities came as a package," said Bingham, director of the Perception/Action Lab at IU. Language is special, and we acquire it very rapidly when young. Recent theories and evidence suggest that perceptual biases in auditory perception channel auditory development, so that we become attuned to the relevant acoustic units for speech. Our work on the size-weight illusion is now suggesting that a similar bias exists in object perception that corresponds to human readiness to acquire throwing skills."

Bingham and Zhu, who completed his doctorate in the Department of Kinesiology at IU's School of Health, Physical Education and Recreation, put their theory to the test, recruiting 12 adult men and women to perform various tests related to perception, the size-weight illusion and throwing prowess.

Another way of stating the size-weight illusion is that for someone to perceive that two objects -- one larger than the other -- weigh the same, the larger object must weigh significantly more than the smaller object. Their study findings show that skilled throwers use this illusion of 'equal felt' heaviness to select objects that they are able to throw to the farthest, maximum distance. This, says Bingham, suggests the phenomenon is not actually an illusion but instead a "highly useful and accurate perception."

Neanderthals, which co-existed with Homo sapiens long ago, lacked the more developed cerebellum and posterior parietal cortex.

"These brain structures have recently been found to distinguish Homo sapiens from Neanderthals," Bingham said. "It is possible that this is what enabled us to beat out Neanderthals, who otherwise had the larger brains."