Common food dye may hold promise in treating spinal cord injury

A common food additive that gives M&Ms and Gatorade their blue tint may offer promise for preventing the additional – and serious – secondary damage that immediately follows a traumatic injury to the spinal cord. In an article published online today in the Proceedings of the National Academy of Sciences, researchers report that the compound Brilliant Blue G (BBG) stops the cascade of molecular events that cause secondary damage to the spinal cord in the hours following a spinal cord injury, an injury known to expand the injured area in the spinal cord and permanently worsen the paralysis for patients.

This research builds on landmark laboratory findings first reported five years ago by researchers at the University of Rochester Medical Center. In the August 2004 cover story of Nature Medicine, scientists detailed how ATP, the vital energy source that keeps our body's cells alive, quickly pours into the area surrounding a spinal cord injury shortly after it occurs, and paradoxically kills off what are otherwise healthy and uninjured cells.

This surprising discovery marked a milestone in establishing how secondary injury occurs in spinal cord patients. It also laid out a potential way to stop secondary spinal injury, by using oxidized ATP, a compound known to block ATP's effects. Rats with damaged spinal cords who received an injection of oxidized ATP were shown to recover much of their limb function, to the point of being able to walk again, ambulating effectively if not gracefully.

Now, scientists detail the clearing of yet another hurdle in moving this research closer from bench to bedside by successfully identifying a compound that could be administered systemically to achieve the same benefit. Previously, the team needed to inject a compound directly into the injured spinal cord area to achieve its results.

"While we achieved great results when oxidized ATP was injected directly into the spinal cord, this method would not be practical for use with spinal cord-injured patients," said lead researcher Maiken Nedergaard, M.D., D.M.Sc., professor of Neurosurgery and director of the Center for Translational Neuromedicine at the University of Rochester Medical Center. "First, no one wants to put a needle into a spinal cord that has just been severely injured, so we knew we needed to find another way to quickly deliver an agent that would stop ATP from killing healthy motor neurons. Second, the compound we initially used, oxidized ATP, cannot be injected into the bloodstream because of its dangerous side effects."

Nedergaard cautions that while this body of work offers a promising new way of treating spinal cord injury, it is still years away from possible application in patients. In addition, any potential treatments would only be helpful to people who have just suffered a spinal cord injury, not for patients whose injury is more than a day old. Just as clot-busting agents can help patients who have had a stroke or heart attack who get to an emergency room within a few hours, so a compound that could stem the damage from ATP might help patients who have had a spinal cord injury and are treated immediately.

Too Much of a Good Thing

While ATP is usually considered to be helpful to our bodies – after all, it's the main source of energy for all of our body's cells – Nedergaard was the first to uncover its darker side in the spinal cord. Immediately after a spinal cord injury occurs, ATP surges to the damaged area, at levels hundreds of times higher than normal. It is this glut of ATP that over-stimulates neurons and causes them to die from metabolic stress.

Neurons in the spinal cord are so susceptible to ATP because of a molecule known as "the death receptor." Scientists know that the receptor – called P2X7 – plays a role in regulating the deaths of immune cells such as macrophages, but in 2004, Nedergaard's team discovered that P2X7 also is carried in abundance by neurons in the spinal cord. P2X7 allows ATP to latch onto motor neurons and send them the flood of signals that cause their deaths, worsening the spinal cord injury and resulting paralysis.

So the team set its sights on finding a compound that not only would prevent ATP from attaching to P2X7, but could be delivered intravenously. In a fluke, Nedergaard discovered that BBG, a known P2X7R antagonist, is both structurally and functionally equivalent to the commonly used FD&C blue dye No. 1. Approved by the Food and Drug Administration as a food additive in 1982, more than 1 million pounds of this dye are consumed yearly in the U.S.; each day, the average American ingests 16 mgs. of FD&C blue dye No. 1.

"Because BBG is so similar to this commonly used blue food dye, we felt that if it had the same potency in stopping the secondary injury as oxidized ATP, but with none of its side effects, then it might be great potential treatment for cord injury," Nedergaard said.

The team was not disappointed. An intravenous injection of BBG proved to significantly reduce secondary injury in spinal cord-injured rats, who improved to the point of being able to walk, though with a limp. Rats that had not received the BBG solution never regained the ability to walk. There was one side effect: Rats who were injected with BBG temporarily had a blue tinge to their skin.

Nedergaard's long-time collaborator on this and other projects, chair of the University of Rochester Department of Neurology Steven Goldman, M.D., Ph.D., adds, "We have no effective treatment now for patients who have an acute spinal cord injury. Our hope is that this work will lead to a practical, safe agent that can be given to patients shortly after injury, for the purpose of decreasing the secondary damage that we have to otherwise expect."

Nedergaard and Goldman believe that further laboratory testing will be needed to test the safety of BBG and related agents before human clinical trials could begin. Nonetheless, the investigators are optimistic that with sufficient study, strategies like this could yield new treatments for acute spinal cord injuries within the next several years.

Other authors from the University of Rochester Medical Center include Weiguo Peng, Maria L. Cotrina, Xiaoning Han, Hongmei Yu, Lane Bekar, Livnat Blum, Takahiro Takano, and Guo-Feng Tia.

The research was supported by the New York State Spinal Cord Injury program, the Miriam and Sheldon Adelson Medical Research Foundation, and grants from the National Institutes of Health.

Heart failure: Women different than men

Absence of women in clinical trials hinders development of tailored therapy

Striking differences in the risk factors for developing heart failure (HF) and patient prognosis exist between men and women. Men and women may also respond differently to treatment, raising concerns about whether current practices provide the best care and reinforcing the urgency for sex-specific clinical trials for HF, according to a review article published in the August 4, 2009, issue of the Journal of the American College of Cardiology.

"Current practice is to treat heart failure similarly in men and women," said Eileen Hsich, M.D., director of the Women's Heart Failure Clinic at the Cleveland Clinic in Ohio. "Yet, our review of published reports suggests compelling sex differences, not only in terms of how and when heart failure develops, but also possible responses to treatments and how the disease impacts quality of life."

The data show that HF - a life-threatening condition in which the heart cannot pump enough blood throughout the body - affects women at an older age and often with a stronger heart compared to men. Hypertension and valvular disease are more likely the culprits for HF in women, whereas men are more likely to have coronary artery disease (CAD) as the underlying cause. And while women live longer with the disease, they also tend to have lower quality of life than men due to greater physical limitations with exercise, more HF-related hospital stays and depression.

"The reasons why survival is better for women remain unclear, but it may be due to differences in the underlying disease," said Dr. Hsich. "Our findings also raise questions as to whether certain diagnostic tests or criteria need to be changed to better reflect how HF presents in female versus male patients."

For example, "normal" values for brain natriuretic peptide - a biomarker that is being used more frequently to identify patients with symptoms of HF and stratify patients by risk - are higher for women versus men and abnormal values with a BNP > 500 pg/ml may be a stronger predictor of death in women with HF than in men. There is also evidence that sex-specific differences may result when performing a cardiopulmonary stress test, which is often used to evaluate patients for heart transplantation. Women with HF tend to have a better prognosis for any given peak oxygen consumption value when compared to men, yet the cut-off values to determine need for heart transplantation are the same for both sexes. The potential benefits of certain HF therapies both in terms of reducing morbidity and mortality appear to be different among women.

"We found that some of the available medications may not be as effective in women, while other therapies, for example, beta blockers, aldosterone antagonists and pacemakers, may be very beneficial," said Dr. Hsich, although she cautions that these finding should in no way prompt women to deviate from what their doctor recommends.

"We need to remember that the therapy women are receiving must be working because they are living longer," she added. "Still, we need to gain a better understanding of HF in women so that we know whether we are providing the best possible care."

A critical challenge remains enrolling women in clinical trials and inspiring researchers to conduct sex-specific studies.

"This is a disease that affects women just as much as men, yet it remains poorly understood and women are still underrepresented in studies," said Dr. Hsich, adding that major multicenter HF trials in the last decade on average only included 28 percent women. "It is really important for women to speak up and not wait for their doctor to approach them about participating in a clinical trial. In doing so, we can help ensure that future advances in HF treatments are applicable to women and supported by sound research."

Approximately 2.7 million women have HF, which accounts for 35 percent of the total female cardiovascular mortality.

Dr. Hsich and co-author, Ileana Pina, M.D., Case Western Reserve University, performed a systematic review of the literature and also contacted the lead investigators of the major CV trials to request sex-specific data if it was not provided in their original publication. Dr. Hsich reports no conflicts of interest.

Divorce undermines health in ways remarriage doesn't heal

Impact of chronic illness lingers after remarriage

Divorce and widowhood have a lingering, detrimental impact on health, even after a person remarries, research at the University of Chicago and Johns Hopkins University shows.

"Among the currently married, those who have ever been divorced show worse health on all dimensions. Both the divorced and widowed who do not remarry show worse health on all dimensions," said University of Chicago sociologist Linda Waite and co-author of a new study on marriage and health. Waite, the Lucy Flower Professor in Sociology and Director of the Center on Aging at the National Opinion Research Center at the University, conducted the study with Mary Elizabeth Hughes, Assistant Professor at Johns Hopkins' Bloomberg School of Public Health. Their research will be published in the September issue of the Journal of Health and Social Behavior in the article, "Marital Biography and Health Midlife," which was based on a study of 8,652 people aged 51 to 61.

Although a number of studies have looked at the connection between health and marriage, theirs is the first to examine both marital transitions and marital status on a wide range of health dimensions. Based on genetics and other factors, people enter adulthood with a particular "stock" of health, other research has shown. "Each person's experience of marital gain and loss affect this stock of health," Waite said. "For example, the transition to marriage tends to bring an immediate health benefit, in that it improves health behaviors for men and financial well-being for women."

These advantages are enhanced throughout marriage. Divorce or widowhood undermines health because incomes drop, and stress develops over issues such as shared child care.

Among the findings:

* Divorced or widowed people have 20 percent more chronic health conditions such as heart disease, diabetes or cancer than married people. They also have 23 percent more mobility limitations, such as trouble climbing stairs or walking a block.

* People who never married have 12 percent more mobility limitations and 13 percent more depressive symptoms, but report no difference in the number of chronic health conditions from married people.

* People who remarried have 12 percent more chronic conditions and 19 percent more mobility limitations, but no more depressive symptoms, than those who are continuously married.

The impacts of marriage, divorce and remarriage on health are based on the ways in which the various illnesses develop and heal, Waite said.

"Some health situations, like depression, seem to respond both quickly and strongly to changes in current conditions," she said. "In contrast, conditions such as diabetes and heart disease develop slowly over a substantial period and show the impact of past experiences, which is why health is undermined by divorce or widowhood, even when a person remarries."

Earliest animals lived in a lake environment, research shows

UC Riverside-led study raises questions about where early animals were living

RIVERSIDE, Calif. – Evidence for life on Earth stretches back billions of years, with simple single-celled organisms like bacteria dominating the record. When multi-celled animal life appeared on the planet after 3 billion years of single cell organisms, animals diversified rapidly.

Conventional wisdom has it that animal evolution began in the ocean, with animal life adapting much later in Earth history to terrestrial environments.

Now a UC Riverside-led team of researchers studying ancient rock samples in South China has found that the first animal fossils in the paleontological record are preserved in ancient lake deposits, not marine sediments as commonly assumed.

"We know that life in the oceans is very different from life in lakes, and, at least in the modern world, the oceans are far more stable and consistent environments compared to lakes which tend to be short-lived features relative to, say, rates of evolution," said Martin Kennedy, a professor of geology in the Department of Earth Sciences who participated in the research. "Thus it is surprising that the first evidence of animals we find is associated with lakes, a far more variable environment than the ocean."

The study, published in the July 27-31 online edition of the Proceedings of the National Academy of Sciences, raises questions such as what aspects of the Earth's environment changed to enable animal evolution.

In their research, the authors focused on South China's Doushantuo Formation, one of the oldest fossil beds that houses highly preserved fossils dated to about 600 million years ago. These beds have no adult fossils. Instead, many of the fossils appear as bundles of cells interpreted to be animal embryos.

"Our first unusual finding in this region was the abundance of a clay mineral called smectite," said lead author Tom Bristow, who worked in Kennedy's lab. "In rocks of this age, smectite is normally transformed into other types of clay. The smectite in these South China rocks, however, underwent no such transformation and have a special chemistry that, for the smectite to form, requires specific conditions in the water – conditions commonly found in salty, alkaline lakes."

The researchers' work involved collecting hundreds of rock samples from several localities in South China, carrying out mineralogical analysis using X-ray diffraction, and collecting and analyzing other types of geochemical data.

"All our analyses show that the rocks' minerals and geochemistry are not compatible with deposition in seawater," Bristow said. "Moreover, we found smectite in only some locations in South China, and not uniformly as one would expect for marine deposits. This was an important indicator that the rocks hosting the fossils were not marine in origin. Taken together, several lines of evidence indicated to us that these early animals lived in a lake environment."

Bristow noted that the new research gives scientists a glimpse into where some of the early animals lived and what the environmental conditions were like for them – important information for addressing the broader questions of how and why animals appeared when they did.

"It is most unexpected that these first fossils do not come from marine sediments," Kennedy said. "It is possible, too, that similarly aged or older organisms also existed in marine environments and we have not found them. But at the very least our work shows that the range of early animal habitats was far more expansive than presently assumed and raises the exciting possibility that animal evolution first occurred in lakes and is tied to some environmental aspect unique to lake environments. Furthermore, because lakes are of limited size and not connected to each other, there may have been significant parallel evolution of organisms. Now we must wait and see if similar fossils are found in marine sediments."

Kennedy, who directs the Graduate Program in Global Climate and Environmental Change at UCR, and Bristow were joined in the study by Mary Droser and Arkadiusz Derkowski of UCR; Ganqing Jiang of the University of Nevada, Las Vegas; and Robert Creaser of the University of Alberta, Edmonton, Canada.

Cardiothoracic surgeons projected to be in short supply by 2025

Study highlights:

* Within the next 15 years, the United States faces a severe shortage of cardiothoracic surgeons – possibly resulting in diminished quality of care and delayed care for heart and lung surgery patients.

* Health and population trends could result in a 46 percent increased demand for cardiothoracic surgeons by 2025, while the supply of these surgeons is projected to shrink by at least 21 percent during the same time.

DALLAS, – Health and population trends could increase demand for cardiothoracic surgeons in the United States far greater than the supply – diminishing and delaying care, according to a report in Circulation: Journal of the American Heart Association.

A study undertaken by the Association of American Medical Colleges' (AAMC) Center for Workforce Studies found that the demand for cardiothoracic surgery services is projected to increase by 46 percent by 2025 (compared to 2005), while the supply of these surgeons is expected to decrease 21 percent during that period.

The supply for cardiothoracic surgeons (physicians specially trained in surgeries of the heart and chest) is already dwindling, said Irving L. Kron, M.D., senior author of the study and Chair of Surgery and professor in the division of thoracic and cardiovascular surgery at the University of Virginia Health Sciences Center.

"The number of active cardiothoracic surgeons has fallen for the first time in 20 years," Kron said. "In 2007, 33 percent of available thoracic surgery fellowship positions went unfilled in the National Resident Matching Program. Surveys of residents in training in cardiothoracic surgery indicated that many were having difficulty finding employment after completing five years of general surgery training, followed by two years of a cardiothoracic surgery fellowship."

This could be, in part, because use of coronary artery bypass grafting (CABG) – the most common procedure performed by cardiothoracic surgeons – is declining (down 28 percent from 1997-2004). Meanwhile cardiac stent placement, performed by cardiologists rather than surgeons, is increasing (up 121 percent from 1997-2004.)

"Stenting is a much less invasive procedure than open-heart surgery and can be performed by an interventional cardiologist," he said. "However, it is not always a suitable substitute for CABG. Furthermore,

patients with stents may ultimately end up needing CABG down the road, although there is still limited data on long-term outcomes."

The elderly are far more likely to need heart surgery, despite the decrease in CABG, thus increasing the need for cardiothoracic surgeons as the population continues to age.

Kron and colleagues projected the supply and demand for cardiothoracic surgeons by analyzing the general population, as well as workplace shifts in the cardiothoracic surgeon population. They used simulation models to predict what might happen in various scenarios.

"The U.S. population is growing by 25 million a decade and the over-65 population is projected to double between 2000 and 2030," Kron said. "Even if there were an immediate increase in the number of residents entering training, we would likely still see an overall decline in the supply of cardiothoracic surgeons over the next 20 years."

A shortage of these specialists could result in patients experiencing significant waiting time before getting needed surgeries. This could potentially lead to unnecessary complications and deaths.

In general, population groups with less access to medical care, especially early care, tend to have poorer health outcomes; so, these populations could suffer most. And though there are some non-surgical options for treating cardiac patients, the shortfall of cardiothoracic surgeons comes at a time when cardiologists will likely be in short supply as well, according to the paper.

The impending shortage of cardiothoracic surgeons is an "important threat," said Timothy Gardner, M.D., immediate past president of the American Heart Association, a cardiac thoracic surgeon and Medical Director for the Center for Heart and Vascular Health, Christiana Care Health System, Newark, Del.

"It is the American Heart Association's mission to promote the cardiovascular health of the population and effectively treat people with cardiac conditions," he said. "If the supply of key specialists, such as heart surgeons, declines, that could impact the health of the population and physicians' abilities to effectively treat people with heart disease."

Co-authors include: Atul Grover, M.D., Ph.D.; Karyn Gorman, M.S.P.A.; Tim Dall, M.S.; Richard Jonas, M.D.; Bruce Lytle, M.D.; Richard Shemin, M.D.; and Douglas Wood, M.D. Individual author disclosures can be found on the manuscript.

The American Association for Thoracic Surgery and the Society of Thoracic Surgeons funded the study.

Smart machines: What's the worst that could happen?

* 15:38 27 July 2009 **by MacGregor Campbell**

An invasion led by artificially intelligent machines. Conscious computers. A smartphone virus so smart that it can start mimicking you. You might think that such scenarios are laughably futuristic, but some of the world's leading artificial intelligence (AI) researchers are concerned enough about the potential impact of advances in AI that they have been discussing the risks over the past year. Now they have revealed their conclusions.

Until now, research in artificial intelligence has been mainly occupied by myriad basic challenges that have turned out to be very complex, such as teaching machines to distinguish between everyday objects. Human-level artificial intelligence or self-evolving machines were seen as long-term, abstract goals not yet ready for serious consideration.

Now, for the first time, a panel of 25 AI scientists, roboticists, and ethical and legal scholars has been convened to address these issues, under the auspices of the Association for the Advancement of Artificial Intelligence (AAAI) in Menlo Park, California. It looked at the feasibility and ramifications of seemingly far-fetched ideas, such as the possibility of the internet becoming self-aware.

The panel drew inspiration from the 1975 Asilomar Conference on Recombinant DNA in California, in which over 140 biologists, physicians, and lawyers considered the possibilities and dangers of the then emerging technology for creating DNA sequences that did not exist in nature. Delegates at that conference foresaw that genetic engineering would become widespread, even though practical applications – such as growing genetically modified crops – had not yet been developed.

Unlike recombinant DNA in 1975, however, AI is already out in the world. Robots like Roombas and Scoobas help with the mundane chores of vacuuming and mopping, while decision-making devices are assisting in complex, sometimes life-and-death situations. For example, Poseidon Technologies, sells AI systems that help lifeguards identify when a person is drowning in a swimming pool, and Microsoft's Clearflow system helps drivers pick the best route by analysing traffic behaviour.

At the moment such systems only advise or assist humans, but the AAAI panel warns that the day is not far off when machines could have far greater ability to make and execute decisions on their own, albeit within a narrow range of expertise. As such AI systems become more commonplace, what breakthroughs can we reasonably expect, and what effects will they have on society? What's more, what precautions should we be taking?

These are among the many questions that the panel tackled, under the chairmanship of Eric Horvitz, president of the AAAI and senior researcher with Microsoft Research. The group began meeting by phone and teleconference in mid-2008, then in February this year its members gathered at Asilomar, a quiet town on the north California coast, for a weekend to debate and seek consensus. They presented their initial findings at the International Joint Conference for Artificial Intelligence (IJCAI) in Pasadena, California, on 15 July.

Panel members told IJCAI that they unanimously agreed that creating human-level artificial intelligence – a system capable of expertise across a range of domains – is possible in principle, but disagreed as to when such a breakthrough might occur, with estimates varying wildly between 20 and 1000 years.

Panel member Tom Dietterich of Oregon State University in Corvallis pointed out that much of today's AI research is not aimed at building a general human-level AI system, but rather focuses on "idiot-savants" systems good at tasks in a very narrow range of application, such as mathematics.

The panel discussed at length the idea of an AI "singularity" – a runaway chain reaction of machines capable of building ever-better machines. While admitting that it was theoretically possible, most members were skeptical that such an exponential AI explosion would occur in the foreseeable future, given the lack of projects today that could lead to systems capable of improving upon themselves. "Perhaps the singularity is not the biggest of our worries," said Dietterich.

A more realistic short-term concern is the possibility of malware that can mimic the digital behavior of humans. According to the panel, identity thieves might feasibly plant a virus on a person's smartphone that would silently monitor their text messages, email, voice, diary and bank details. The virus could then use these to impersonate that individual with little or no external guidance from the thieves. Most researchers think that they can develop such a virus. "If we could do it, they could," said Tom Mitchell of Carnegie Mellon University in Pittsburgh, Pennsylvania, referring to organised crime syndicates.

Peter Szolovits, an AI researcher at the Massachusetts Institute of Technology, who was not on the panel, agrees that common everyday computer systems such as smartphones have layers of complexity that could lead to unintended consequences or allow malicious exploitation. "There are a few thousand lines of code running on my cell phone and I sure as hell haven't verified all of them," he says.

"These are potentially powerful technologies that could be used in good ways and not so good ways," says Horvitz, and cautions that besides the threat posed by malware, we are close to creating systems so complex and opaque that we don't understand them.

Given such possibilities, "what's the responsibility of an AI researcher?" says Bart Selman of Cornell, co-chair of the panel. "We're starting to think about it."

At least for now we can rest easy on one score. The panel concluded that the internet is not about to become self-aware.

How the pathology of Parkinson's disease spreads

Neuron-to-neuron transmission of a-synuclein may cause alpha-synuclein aggregates to propagate

Accumulation of the synaptic protein alpha-synuclein, resulting in the formation of aggregates called Lewy bodies in the brain, is a hallmark of Parkinson's and other related neurodegenerative diseases. This pathology appears to spread throughout the brain as the disease progresses. Now, researchers at the University of California, San Diego School of Medicine and Konkuk University in Seoul, South Korea, have described how this mechanism works. Their findings – the first to show neuron-to-neuron transmission of alpha-synuclein – will appear in the Proceedings of the National Academy of Sciences (PNAS) on July 29.

"The discovery of cell-to-cell transmission of this protein may explain how alpha-synuclein aggregates can pass to new, healthy cells," said first author Paula Desplats, project scientist in UC San Diego's Department of Neurosciences. "We demonstrated how alpha-synuclein is taken up by neighboring cells, including grafted neuronal precursor cells, a mechanism that may cause Lewy bodies to spread to different brain structures."

This insight will impact research into stem cell therapy for Parkinson's disease. "Our findings indicate that the stem cells used to replace lost or damaged cells in the brains of Parkinson's disease patients are also susceptible to degeneration," said Eliezer Masliah, MD, professor of neurosciences and pathology at UC San Diego School of Medicine. "Knowledge of the molecular basis of the intercellular transmission of alpha-synuclein may result in improved stem-cell based therapies with long-lasting benefits, by preventing the grafted cells to uptake α-synuclein or by making them more efficient in clearing the accumulated alpha-synuclein ."

In a large proportion of Parkinson's disease cases, the aggregation of alpha-synuclein progresses in a predictable pattern - from the lower brainstem, into the limbic system and eventually to the neocortex, the part of the brain responsible for higher level cognitive functions. The hypothesis of disease progression by neuron-to-neuron transmission of alpha-synuclein that encouraged this study was supported by findings of two separate

reports in 2008. In these studies, autopsies of deceased Parkinson's patients who had received implants of therapeutic fetal neurons 11 to 16 years prior revealed that alpha-synuclein had propagated to the transplanted neurons.

Collaborating with South Korean researcher Seung-Jae Lee, the UC San Diego researchers first looked at neural precursor cells in culture, co-culturing them with neuronal cells expressing alpha-synuclein . After 24 to 48 hours, the aggregated alpha-synuclein was evident in the precursor cells – results suggesting cell-to-cell transmission. Using specific inhibitors, the research team also discovered that alpha-synuclein is transmitted via endocytosis, the normal process by which cells absorb proteins from the extracellular media by engulfing them within their cell membrane. Blockage of the endocytic pathway resulted in lesser accumulation of alpha-synuclein

Additionally, the researchers found that failure of the quality-control systems of the cell contributes to the observed accumulation of alpha-synuclein in recipient cells. This is due to inhibited activity of cell particles called lysosomes, which would usually degrade and remove aggregates – resulting in their increased formation.

Next, the team tested to determine if alpha-synuclein could be transmitted directly from host to grafted cells in a mouse model of Parkinson's disease. Brains of the mouse model were grafted with fresh, healthy stem cells. Within four weeks, cells containing Lewy body-like masses were quite common, supporting the cell-to cell transmission mechanism.

Contributors to the study included co-first author He-Jin Lee and Eun-Jin Bae of Konkuk University in Seoul, South Korea; and Christina Patrick, Edward Rockenstein, Leslie Crews and Brian Spencer of the UC San Diego School of Medicine. The research was supported by the Brain Research Center of the 21st Century Frontier Research Program, funded by the Ministry of Education, Science and Technology; the Diseases Network Research Program of the Ministry of Education, Science and Technology; the Korea Science and Engineering Foundation, funded by the Korean government, and grants from the U.S. National Institutes of Health.

ISU researchers find possible treatment for Spinal Muscular Atrophy

AMES, Iowa - Spinal Muscular Atrophy is the second-leading genetic cause of infant mortality in the world.

Ravindra Singh, associate professor in biomedical sciences at Iowa State University's College of Veterinary Medicine, would like to see Spinal Muscular Atrophy lose its high ranking and even slide off the list altogether.

Most Spinal Muscular Atrophy sufferers - more than 95 percent - have a mutated or deleted gene called Survival Motor Neuron 1 (SMN1) that doesn't correctly do its job of creating functional SMN proteins.

Singh's solution is to replace that poor-performing gene with another gene.

Humans need a certain level of SMN protein to ward off Spinal Muscular Atrophy. When SMN1 fails to create functioning proteins, Spinal Muscular Atrophy is the result.

There is a gene already in humans that looks very much like SMN1, so much so that it's called SMN2. The SMN2 gene doesn't seem to serve any function that researchers can identify.

Singh has discovered a way of using SMN2 to produce the working SMN protein. When SMN2 makes enough SMN, it compensates for the mutated or malfunctioning SMN1 gene.

All proteins in human bodies are made by copying genes. This copy is called pre-mRNA. Pre-mRNA then becomes mRNA by splicing out certain parts of the sequence that are non-coding, meaning they don't help the function of the gene. These non-coding portions of the pre-mRNA are called intronic sequences, sometimes referred to as junk sequence because it is originally copied from junk DNA.

SMN2 normally doesn't produce normal protein because of the presence of a specific intronic sequence in the gene or DNA.

To make SMN2 behave as SMN1, Singh has introduced a small antisense oligonucleotide that blocks this specific intronic sequence.

When the intronic sequence is blocked, SMN2 produces normal proteins and acts, in effect, like SMN1.

"The significance of our work is that we have this stuff called junk DNA in SMN2," said Singh. "We found that we could get SNM2 to behave as SMN1 by introducing a small oligonucleotide. It is a very simple experiment if you think about it."

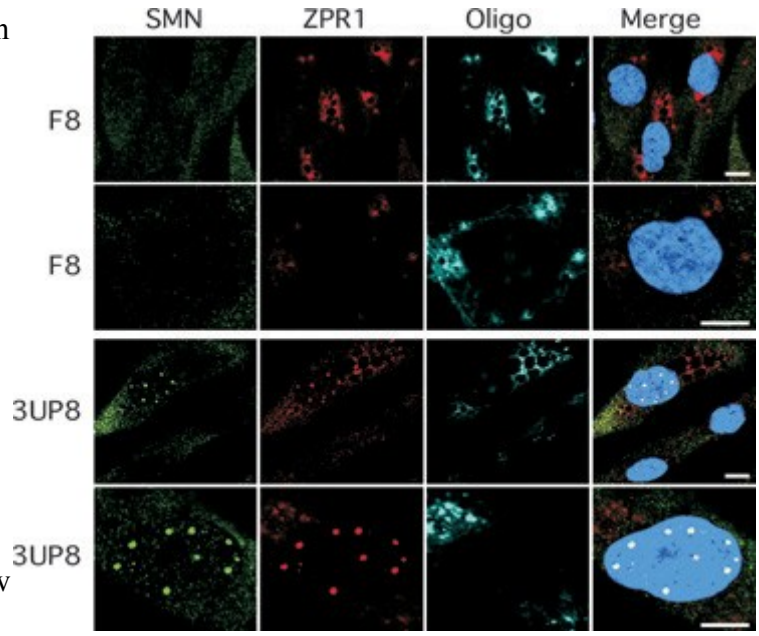
The resulting proteins are normal just like a regular cell - free from Spinal Muscular Atrophy.

"Our cells are healthy and survive," he said. "From that point of view, this is a major achievement."

Singh, along with his team Natalia Singh and Maria Shishimorova, both of Iowa State University's biomedical services department; Lu Cheng Cao, University of Massachusetts Medical School, Worcester; and Laxman Gangwani, Medical College of Georgia, Augusta, have their research highlighted as the cover story on this month's issue of the journal RNA Biology. Their research is the most downloaded story on the RNA Biology page of the Web site Landes Bioscience.

Spinal Muscular Atrophy affects 1 in 6,000 to 1 in 10,000 children born every year. One in 40 people are carriers of the disease - they don't have the symptoms, but could pass the disease to their children. Most children born with the most severe type of SMA die within two years. Using this junk sequence in SMN2 to restore the high levels of functional SMN protein could eliminate Spinal Muscular Atrophy caused by deletion or mutation in SMN1. Singh believes this technology could also work treating other diseases.

"We know that Parkinson's disease, Alzheimer's disease, cystic fibrosis, multiple sclerosis and cancer all come from genes that are aberrantly spliced. If this is a model disease, meaning we succeed in treating Spinal Muscular Atrophy, we will know how to correct splicing of other genes in other diseases," he said.



A short and smart oligonucleotide: A short antisense oligonucleotide (3UP8) targeting a specific intronic sequence corrects aberrant splicing of Survival Motor Neuron 2 (SMN2) and restores high levels of functional SMN protein in patient cells of spinal muscular atrophy (SMA). Prominent green dots (see in "SMN section") represent well-organized SMN bodies (gems) in the nucleus of the 3UP8-treated cells (bottom two panels). Gem contains another protein ZPR1 (see red dots in "ZPR1 section") that co-localizes with SMN protein in the nucleus of the cell (see white dots in "Merge section").

The control oligonucleotide (F8) that targets an unrelated sequence had no effect on SMN or ZPR1 levels in patient cells (top two panels). Cell were transfected with the same amount of antisense oligonucleotide (compare the color intensity in "Oligo section").

Comets, not asteroids, to blame for moon's scarred face

* 19:28 27 July 2009 **by Hazel Muir**

Icy comets - not rocky asteroids - launched a dramatic assault on the Earth and moon around 3.85 billion years ago, a new study of ancient rocks in Greenland suggests. The work suggests much of Earth's water could have been brought to the planet by comets.

"We can see craters on the moon's surface with the naked eye, but nobody actually knew what caused them - was it rocks, was it iron, was it ice?" says Uffe Gråe Jørgensen, an astronomer at the Niels Bohr Institute in Copenhagen, Denmark. "It's exciting to find signs that it was actually ice."

Evidence suggests that the Earth and moon had both formed around 4.5 billion years ago. But almost all the craters on the moon date to a later period, the "Late Heavy Bombardment" 3.8 to 3.9 billion years ago, when around 100 million billion tonnes of rock or ice crashed onto the lunar surface. The Earth would have been pummelled by debris at the same time, although plate tectonics on our restless planet have since erased the scars.

To find out whether asteroids or comets were the main culprits for the bombardment, Jørgensen decided to measure levels of the element iridium in ancient terrestrial rocks. Iridium is rare on the Earth's surface because almost all of it bound to iron and sank into the Earth's core soon after the planet had formed. But iridium is relatively common in comets and meteorites.

Rock or ice

His team calculated the amount of iridium that asteroids would leave on the Earth and moon compared to comets. Because comets have more volatile elements and higher impact speeds due to their more elongated orbits around the sun, they would create giant plumes on impact, allowing more iridium to escape into space than during asteroid impacts.

The team predicted that asteroid bombardment would leave iridium levels of 18,000 and 10,000 parts per trillion in rocks on the Earth and moon respectively, while the same figures for comet bombardment would be about 130 and 10.

Ancient moon rocks returned by NASA's Apollo missions have already confirmed that the lunar iridium levels are 10 parts per trillion or less. To find out the terrestrial value, Jørgensen's team sampled some of the world's oldest rocks from Greenland, aged 3.8 billion years, and asked a Japanese laboratory to assess their iridium levels more accurately than ever before. They contained iridium levels of 150 parts per trillion.

That strongly suggests comets, rather than asteroids, caused the violent bombardment.

Giant plumes

If so, Jørgensen's team calculates that around 3400 tonnes of icy comet material fell on each square metre of the Earth. About half the comet material would ricochet back into space in giant plumes, leaving behind roughly a billion cubic kilometres of cometary water in total.

That is a similar amount to that in the Earth's oceans today, although it is not clear whether there was already water on the planet due to chemical reactions on the early Earth (see Earth's water brewed at home, not in space).

Michael Mumma, a comet expert at NASA's Goddard Space Flight Center in Maryland who was not involved in the research, says the new report is interesting: "The paper is certain to stimulate lively debate."

Journal reference: Icarus (10.1016/j.icarus.2009.07.015)

After dinosaurs, mammals rise but their genomes get smaller

BLOOMINGTON, Ind. - Evidence buried in the chromosomes of animals and plants strongly suggests only one group - mammals - have seen their genomes shrink after the dinosaurs' extinction. What's more, that trend continues today, say Indiana University Bloomington scientists in the first issue of a new journal, Genome Biology and Evolution.

The scientists' finding might seem counter-intuitive, given that the last 65 million years have seen mammals expand in diversity and number, not to mention dominance in a wide variety of ecological roles. But it is precisely their success in numbers that could have led to the contraction of their genomes.

"Larger population sizes make natural selection more efficient," said IU Bloomington evolutionary biologist Michael Lynch, who led the study. "If we are correct, we have shown how to bring ancient genomic information together with the paleontological record to learn more about the past."

And the present. Lynch says the data he and his colleagues analyzed suggest human genomes are still undergoing a contraction - though you shouldn't expect to see noticeable changes in our chromosomes for a few million years yet.

Lynch's group examined the genomes of seven mammals, eight non-mammalian animals and three plants, specifically with regard for the long terminal repeat (LTR) sequences of transposable elements, a curious sort of "jumping" genetic sequence initially dropped into genomes by viruses. IU School of Informatics (Bloomington) bioinformaticians Mina Rho and Haixu Tang oversaw the survey of mammalian and non-mammalian genomes.

Transposable elements often lose their functionality soon after insertion but nevertheless are disturbingly common. In the human genome, for example, transposable elements constitute as much as 45 percent of an individual's total DNA. Long terminal repeat sequences, part of that figure, make up about 8 percent of humans' total DNA.

LTRs come in a range of sizes and ages, and it is the age distribution of LTRs that interested Lynch and his colleagues.

"This study started out as independent observations in the literature," Lynch said. "The data we saw suggested a bulge in age distribution of transposable elements in humans and mouse."

Left enough time, Lynch says, transposable elements are eventually lost from the genome, sometimes by accident and sometimes, perhaps, as the result of natural selection against excess DNA. An LTR is far more likely to survive a few years of cell divisions - and the chance of obliteration via a DNA replication error - than 10 million years of cell divisions. Plotting the full range of 17 species' LTRs, young and old, Lynch and his colleagues usually saw a descending curve with lots of new transposable elements and a dramatic drop-off in the number of older elements.

But not in most mammals. In humans, macaques, cows, dogs and mouse, Lynch's group observed a hill-shaped curve, with a peak of middle-aged LTRs and drop-offs both in the number of older and younger LTRs. The shape of the curve is consistent with previously published data for other types of so-called "junk" DNA elements.

The depressed numbers of very young LTRs, Lynch says, strongly suggests a contraction in overall genome sizes of the lineages of the mammals the scientists studied. That could come about in one of two ways, he says. One possibility is an increase in the efficiency of natural selection that accompanies population growth.

"We think that's the most likely explanation," Lynch said. "Another possibility is that natural selection was just stronger, but we doubt it. For that to be the case, natural selection would have to act in the same way on several lineages around the globe simultaneously."

Mo Zhou, Xiang Gao and Sun Kim also contributed to the report. It was funded with grants from the National Institutes of Health, the National Science Foundation and the Indiana METACyt Initiative, an Indiana University program seeded by the Lilly Foundation.

A Twisted Ankle Isn't Just a Simple Sprain

By JANE E. BRODY

A sprained ankle is one of the most common joint injuries, prompting many people to consider it "just a sprain" and not treat it with the respect it deserves. The too-common consequence of this neglect is a lasting weakness, an unstable joint and repeated sprains.

Given that some 25,000 ankle sprains occur each day in the United States, it is worth knowing how they can be prevented and how they should be treated.

I suffered two memorable ankle sprains, and although I did better with the second than the first, in neither case did I do everything right.

The first occurred 40 years ago when I was nine months pregnant with twins. I twisted my ankle stepping on an uneven surface in my backyard. The pain subsided in a few minutes, and I did nothing about it. Nothing, that is, until it began to swell and throb hours later and I couldn't walk. I was not a pretty sight hobbling to the doctor using my husband as a crutch.

The second occurred about two decades ago, when I turned my ankle coming down the stairs of a commuter plane. This time the acute pain was so severe I had to be carried into the airport, where a wheelchair and ice packs were provided. On my connecting four-hour flight, I was given a three-seat row where I could keep my ankle elevated and periodically iced. I slept that night with the pillows under my foot. The next morning, the pain was gone and I went jogging.

The two mistakes: My first injury should have been treated immediately, with rest, ice and elevation and an elastic bandage to keep down the swelling; with the second, I had no business running on that ankle less than 12 hours after the injury.

No Quick Fix

I now know that I was lucky not to have ended up with a chronically unstable ankle after either of these episodes. Ankle sprains are so often mistreated or not treated at all, experts say, that they have the highest recurrence rate of any joint injury and often result in chronic symptoms.

Last month at the National Athletic Trainers' Association annual meeting in San Antonio, experts responsible for the ankle health of college athletes reviewed research evidence for various methods believed to help prevent recurrent ankle sprains. I suspect that few athletes, whether professional, intramural or recreational, will like the bottom line: ankle sprains usually need more rehabilitation and take longer to heal than most people allow for.

Undertreatment means that "30 to 40 percent of people with simple ankle sprains develop chronic long-term joint pathology," said one presenter, Tricia Hubbard, the undergraduate athletic training director at the University of North Carolina in Charlotte.

"Most research is showing that with any ankle sprain, the ankle should be immediately immobilized to protect the joint and allow the injured ligaments to heal," Dr. Hubbard said in an interview. "At least a week for the simplest sprain, 10 to 14 days for a moderate sprain and four to six weeks for more severe sprains."

Yet coaches, like most people, she said, "tend to think, 'It's just a sprain, you'll be fine' and they tape the ankle and ice it and the player is back on the field in a few days." Of course, players want to play, whatever their level, so they rarely question the wisdom of such a quick turnaround.

"Lack of pain is not always the best indicator that it's safe to resume activity," Dr. Hubbard said. "The pain of an ankle sprain can subside fairly quickly, but that does not mean the injured ligaments have healed."

A Vulnerable Joint

The ankle, which joins the lower leg bones to the foot, is held together by bands of elastic fibers called ligaments. A sprain results when one or more ligaments is stretched beyond its normal range. In a severe sprain, the elastic fibers tear partly or completely.

Sprains occur when the foot turns in or out to an abnormal degree relative to the ankle. Common causes include stepping up or down on an uneven surface, particularly when wearing shoes with platform soles or high heels; stepping wrong off a curb or into a hole; or stepping on an object left in the wrong place.

In athletics, common causes include coming down wrong after a jump shot or rebound; stepping on another player's foot; and having to make quick directional changes, as in tennis, basketball, football and soccer.

As with other such injuries, the recommended first aid for an ankle sprain, to be started as soon as possible after the injury, goes by the acronym RICE:

R for rest, I for ice, C for compression, E for elevation. In other words, get off the foot, wrap it in an Ace-type bandage, raise it higher than the heart and ice it with a cloth-wrapped ice pack applied for 20 minutes once every hour (longer application can cause tissue damage).

This should soon be followed by a visit to a doctor, physical therapist or professional trainer, who should prescribe a period of immobilization of the ankle and rehabilitation exercises. An anti-inflammatory drug may be recommended and crutches provided for a few days, especially if the ankle is too painful to bear weight.

The Healing Process

Immobilization using a brace or cast provides ligaments with the rest they need to heal and reduces the risk of aggravating the injury. Even a complete ligament tear can heal without surgery through proper immobilization. But immobilization should not be overdone and must be followed in a week by exercises that prevent muscle atrophy and stiffness.

During healing, Dr. Hubbard said, “new tissues are laid down, and they need to be aligned with the action of the joint” through proper exercise.

Rehabilitation should include range-of-motion and stretching exercises, strength training and balance training.

Dr. Hubbard said studies had shown that one of the most effective immobilizers is the AirCast Air-Stirrup ankle brace. This inexpensive half-pound device limits motion but can be removed for needed exercises.

Even after an injury has healed, an athlete’s ankle often needs extra protection during physical activities. Studies reviewed by Jay Hertel, an athletic trainer at the University of Virginia, showed that wearing a lace-up ankle brace was more effective than taping the ankle in preventing reinjury.

Of course, preventing injury in the first place is ideal. Athletic trainers emphasize the importance of wearing proper shoes for your chosen activity - shoes that are comfortable, supportive and not worn out.

Dr. Hubbard says women should be very careful in high heels or platform shoes, which she called “an ankle sprain waiting to happen.”

Field Museum scientist describes first vertebrate to live in trees

CHICAGO, IL – In the Late Paleozoic (260 million years ago), long before dinosaurs dominated the Earth, ancient precursors to mammals took to the trees to feed on leaves and live high above predators that prowled the land, Jörg Fröbisch, PhD, a Field Museum paleontologist has concluded. Elongated fingers, an opposable "thumb," and a grasping tail of Suminia getmanovi demonstrate that this small plant-eating synapsid is the earliest known tree-climbing vertebrate

Suminia was relatively small, about 20 inches from its nose to the tip of its tail. The tree-climbing lifestyle of this Paleozoic relative of mammals is particularly important because for the first time in vertebrate evolution it gives access to new food resources high off the ground, and also provides protection from ground-dwelling predators. The evidence for this lifestyle is based on several excellent skulls and more than a dozen exceptionally well preserved, complete skeletons from a single large block of red mudstone that was discovered in central Russia's Kirov region.

Having so many individual specimens, some of mature individuals and some of youngsters, was helpful in providing a complete picture of the animal's skeletal anatomy, said Fröbisch. "It's relatively rare to find several animals locked on a single block," he said. "We have examples of virtually every bone in their bodies."

Finding that vertebrates took to trees so early in Earth's evolution was unexpected. "It's a surprise, but it makes sense," Fröbisch said. "It was a new niche for vertebrates. There was food available and they avoided predators on the ground."

Skeletal reconstruction of the tree-climbing synapsid Suminia getmanovi from the Later Paleozoic (260 million years ago) of Russia. Illustration by Christina Stoppa.

Flesh reconstruction of the tree-climbing synapsid Suminia getmanovi from the Later Paleozoic (260 million years ago) of Russia. Illustration by Christina Stoppa.

The study also provides the first evidence in the fossil record of food partitioning between small climbing and large ground-dwelling plant-eaters and this happens shortly after the establishment of the modern terrestrial ecosystem with large numbers of plant-eaters supporting few top predators. Earlier terrestrial vertebrate communities did not have this modern hierarchy, but instead were composed of various-sized predators and relatively few plant-eaters, with most of the food resources being provided by insects and aquatic organisms.

This research is part of a collaborative study of the first author Dr. Jörg Fröbisch (Department of Geology, Field Museum, Chicago) and Dr. Robert Reisz (Department of Biology, University of Toronto), which will be published on July 29 in the Proceedings of the Royal Society B.

***Funding:** This work was supported by grants from the Government of Canada Awards Program (Full Scholarship), the German Academic Exchange Service (DAAD-Doktorandenstipendium), the University of Toronto, The Field Museum (Visiting Scholarship), and the Deutsche Forschungsgemeinschaft (FR 2457/3-1) to Jörg Fröbisch. and the Natural Sciences and Engineering Research Council of Canada and National Geographic Society to Robert R. Reisz.*

Really?

The Claim: Refrigeration Preserves Nutrients

By ANAHAD O'CONNOR

Leif Parsons

THE FACTS Summer is the time for fresh produce, from farmer's markets to garden harvests. But consumers may not realize that many fruits and vegetables experience rapid losses in their nutritional value when stored for more than a few days.

In part, that is because the produce has usually already spent days in transport and on shelves before you buy it, said Barbara P. Klein, a professor of food science and human nutrition at the University of Illinois at Urbana-Champaign. Once they hit the refrigerator, she added, some fruits and vegetables can lose as much as 50 percent of their vitamin C and other nutrients in the ensuing week, depending on the temperature.

But there are several ways around this. One, look for fresh produce that was locally grown - it has usually traveled shorter distances and is still near its nutritional peak - and try not to stock up on more than a week's supply.

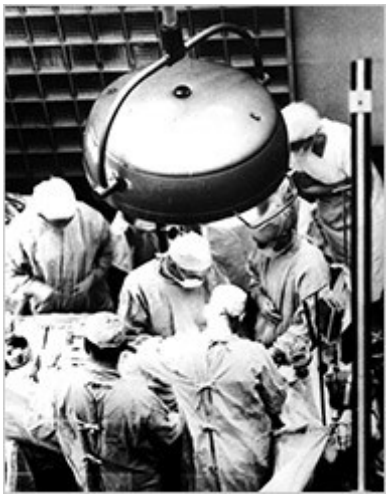
Another option is frozen produce. While frozen fruits and vegetables may lack the flavor and aesthetic appeal of fresh, they are subjected to flash freezing immediately after being picked. That can slow or halt the loss of vitamins and nutrients.

THE BOTTOM LINE Refrigerating produce does not prevent the loss of its nutrients.

First Mention

Kidney Transplant

By NICHOLAS BAKALAR



MAKING HISTORY The first successful kidney transplant was performed at a hospital in Boston. **Brigham and Women's Hospital**

Surgeons tried at least nine times to transplant a human kidney before succeeding. An article written in the spring of 1954 and published in the February 1955 issue of The Journal of Clinical Investigation reported that five of the operations failed immediately, and the rest within 180 days.

Even in animals, the report said, organ transplants had never been successful.

The New York Times mentioned one of these nine operations in an Associated Press report on June 20, 1950. "Kidney Transplant Reported a Success" the headline said, but the report, published three days after the operation, was wrong. The kidney never produced urine effectively and had to be removed nine months later.

The patient, Ruth Tucker, a 49-year-old woman with a genetic illness called polycystic kidney disease, survived with her remaining kidney for another five years.

But then, success.

On Dec. 23, 1954, a team led by Dr. Joseph E. Murray at the Peter Bent Brigham Hospital in Boston transplanted a kidney from a 23-year-old man named Ronald Herrick to his identical twin, Richard, whose kidneys were failing.

This time there were no premature reports. It was not until Nov. 3, 1955, that The Times first mentioned it in an article on Page 33.

"Never before has such a feat of organ transplanting in man been accomplished," Robert K. Plumb wrote. "In no other case in the history of medicine has a human kidney transplant 'taken' and lasted so long. Attempts at transplanting other organs have not succeeded either."

Richard Herrick died in March 1963 after a recurrence of the original kidney disease in his transplanted kidney.

On Oct. 9, 1990, Gina Kolata reported on Page C3 that Dr. Murray had been awarded the Nobel Prize in Physiology or Medicine.

Vital Signs

Regimens: Restrictive Diets May Not Be Appropriate for Children With Autism

By RONI CARYN RABIN

Many parents of autistic children have put their children on strict gluten-free or dairy-free diets, convinced that gastrointestinal problems are an underlying cause of the disorder. But a new study suggests the complicated food regimens may not be warranted.

Researchers at the Mayo Clinic reviewed the medical records of over 100 autistic children over an 18-year period and compared them to more than 200 children without the disorder. The scientists found no differences in the overall frequency of gastrointestinal problems reported by the two groups, though the autistic children suffered more frequently from bouts of constipation and were more likely to be picky eaters who had difficulty gaining weight.

The study, published on Monday in the journal Pediatrics, is the first to look at the incidence of gastrointestinal problems in an autistic population, according to the paper's first author, Dr. Samar H. Ibrahim, a pediatric gastroenterologist at the Mayo Clinic. She suggested that autistic children should only be put on restrictive wheat-free or dairy-free diets after having appropriate diagnostic tests done.

"There is actually no trial that has proven so far that a gluten-free and casein-free diet improves autism," she said. "The diets are not easy to follow and can sometimes cause nutritional deficiencies."

The study found that the vast majority of both autistic and non-autistic children suffered from bouts of common gastrointestinal problems like constipation, diarrhea, abdominal bloating, reflux or vomiting Feeding issues and picky eating were also common. Some 77 percent of autistic children and 72 percent of non-autistic children were affected by one or more of these complaints over the 18-year period.

About 34 percent of the autistic children were affected by constipation, compared to 17.6 percent of the comparison group, while 24.5 percent of the autistic children had feeding issues and were selective in their eating, compared with only 16 percent of the non-autistic group.

But very few of the autistic children had a specific diagnosis of a gastrointestinal disease. Only one autistic child had Crohn's disease, and one had intestinal disaccharidase deficiency and lacked enzymes necessary to digest certain carbohydrates. None suffered from celiac disease, which some reports have linked to autism.

Two of the non-autistic children in the comparison group suffered from lactose intolerance, and one had a milk allergy.

Dr. Ibrahim suggested that the loss of appetite and difficulty gaining weight in autistic children may be related to the use of stimulant medications, which are often prescribed for the condition, and that the constipation may be due to children not consuming enough fiber or drinking enough water.

Scientist at Work: Tucker Childs

Linguist's Preservation Kit Has New Digital Tools

By CHRIS NICHOLSON

TEI, Sierra Leone — Jogue, yipe, simoi are three short words for foods in Kim, a language in Sierra Leone that Tucker Childs has been trying, for the past three years, to write down, record and understand.

Kim is a dying language, and Dr. Childs a field linguist. From his base here in Tei, a small fishing village on the Waanje River, he canoes up the narrow waterways that cut across the river's floodplain, and hikes a few miles inland, to where the last Kim communities remain. Based on recordings taken there, he has devised an alphabet and compiled a dictionary and is finishing a book on the grammar.

Africa has about 2,000 of the world's 6,000 languages. Many are still unwritten, some have yet to be named and many will probably disappear. For centuries, social and economic incentives have been working against Kim and in favor of Mende, a language used widely in the region, until finally, Dr. Childs speculates, the Kim language has been pushed to the verge of extinction.

It used to be that field linguists like Dr. Childs, a scattered corps working against time to salvage the world's endangered tongues - more than 3,000 at last count - scribbled data in smeared notebooks and stored sounds on cassette tapes, destined to rot in boxes. But linguistics has gone digital. Dr. Childs now uses a solid-state recorder, and he has applications that will analyze the elements of a vowel in seconds or compare sounds across languages.

Using Geographic Information Systems, software that translates data into maps, he and his research assistants, Hannah Sarvasy and Ali Turay, pinpoint villages that are not to be found on any official map. "There's a whole bunch of reasons linguists want these languages preserved," Dr. Childs said, "but for me it's more an emotional thing. It's not noblesse oblige, it's capitalist oblige. These people are totally peripheralized."

In its new digital form, this kind of research is more accessible. It allows larger projects to share the world's linguistic heritage with a wider public of teachers and learners, including, when possible, the original speakers.

The aim is not just to salvage, but to revive. Financed by the Hans Rausing Endangered Languages Project and the National Science Foundation, Dr. Childs's recordings will find their way, once his study ends and he returns to his post as a professor at Portland State University in Oregon, to a huge data bank in the School of Oriental and African Studies at the University of London.

The director of the endangered languages archive at S.O.A.S., David Nathan, said the school's Web site, elar.soas.ac.uk, is set to start sharing data at summer's end. "What we're trading in with language documentation is a new genre of stuff which doesn't have any publication channel," he said.

Until now, anyway. The new genre is really a grab bag that includes audio recordings of conversations and folktales, videos of songs and dances, and text transcriptions. But as with most new genres, this one is coming into the world with birthing pains.

Just getting decent recordings can be difficult. The villages of Nyandehun and Mosenten, for example, are roadless, low-tech places. With more elaborate equipment, batteries fail unexpectedly, miles from an electrical outlet. Humidity and dust creep into machines.

Also, some linguists have had trouble mastering the new machines. "For most linguists, audio is just an inconvenience on the way to transcription," Mr. Nathan said. In the past, he added, "the quality would be so bad, it was really just evidence that they had gone there, a talisman that they had gone to the field."

The relationship between linguistics and technology goes deeper than what format the sounds are recorded in. Dr. Childs, who remembers working with computers as large as a room when he was a doctoral student, said that theories of language often shaped themselves to resemble the tools at hand.

In the beginning, he said, linguists imagined that the mind processed language with many rules and little in storage. "What happened over time was that more and more stuff was moved into the lexicon, was listed there, and that sort of paralleled developments in the computer industry of storage getting cheaper," he said.

S.O.A.S. is not alone in trying to document endangered languages. The Max Planck Institute in Nijmegen, the Netherlands, has been operating an archive for 10 years. Dagmar Jung, a linguist in Cologne, Germany, is working with the elders of the Beaver, or Dane-Zaa, tribe in the Canadian provinces of British Columbia and Alberta to gather material and make it accessible through a community portal. "It's there for later generations," Dr. Jung said. "But it's not user-friendly for the moment."

Beaver speakers do have access to some recordings of their songs and stories online. Gary Oker, 49, a former chief of the Dane-Zaa, said putting recordings of elders online was part of a project to take traditional world views and make them part of the present. Dane-Zaa youth were involved throughout the process, from producing the recordings of elders that went online, to using them later as references in school.

Although he saw his language slipping away, he said that as the young people had "taken an oral tradition and documented it in many forms," the contact had made them "prouder of their history and who they are." The stories, he said, helped them learn their identity and how they related to the land.

Because of oil and gas development, Mr. Oker said, "our environment is changing so quickly, we need to capture as much as we can." Even if the language is lost, he said, "the wisdom can be transmitted."

Of course, online resources are useful only to communities with Internet access. Communities without that access, like the Kim, still require books to be printed, and recordings to be copied onto CDs or tapes.

Holding more promise are programs that put electronic dictionaries on mobile phones. James McElvenny, a linguist at the University of Sydney, has led the development of software to help revitalize vanishing languages. Mr. McElvenny has been working with Aboriginal groups like the Dharug of Sydney to give learners, many of them no older than 16, a portable reference that supplies the definition and the sound of words that are otherwise no longer spoken, because Dharug is a dead language.

"A lot of the older members are technophobic," he said, "but the kids are really getting into it."

As for Kim, these efforts may be too late. A language, like a person, usually ages before it dies. Four people have died since Mr. Childs's project began, and the 20 fluent Kim speakers are all over 60.

"People today can't speak Kim because their parents didn't speak it to them," said Fasia Kohlia, one of Kim's best speakers. "Parents used to call their children to breast-feed in Kim — 'kun moga, kun moga, kun moga,' " she said. But when she had children, she called to them in Mende.

Scary music is spookier with eyes shut

* 11:10 28 July 2009 **by Ewen Callaway**

Singers and guitar heroes alike have always employed what you might call the Celine Dion effect – closing your eyes to heighten the emotional impact of music.

Now, neuroscientists have discovered that a brain centre involved in sensing emotion and fear called the amygdala kicks into action when volunteers listen to scary music with eyes closed.

"A lot of time we do like to close our eyes when we listen to music, we feel like this is a more powerful experience," says Talma Hendler, a neuroscientist at Tel Aviv Sourasky Medical Center in Israel, who led the new brain imaging study.

Shutting your eyes heightens people's emotional responses to the outside world, suggests previous research – not to mention everyday experience.

Spooky sounds

To uncover any neural basis for this effect, Hendler's team scanned the brains of 15 volunteers while they listened to film scores – "kind of Hitchcock-like movies," she says – and less emotive keyboard tunes with their eyes open or shut. Hear a scary clip here and a neutral one here.

Sure enough, volunteers rated the eerie-sounding music – laced with staccato strings, ominous trombones, and weird effects – as more emotional than the "elevator music"-like keyboard tunes.

Under the gaze of a functional-MRI scanner, horror film scores elicited significantly more amygdala activity in the brains of volunteers who kept their eyes shut, compared to when they kept eyes open. Participants' brains responded no differently to the neutral music whether their eyes were closed or open.

Threat response

Furthermore, a part of the brain stem that metes out the neurotransmitter noradrenalin in response to threats was more active when volunteers listened to scary music with their eyes closed than open, as was a neocortical brain region known to control emotion – the ventral prefrontal cortex.

None of these changes occurred when volunteers listened to scary music in total darkness, suggesting that temporary blindness – which could certainly heighten fear – doesn't explain the results, Hendler says.

She also thinks her team results aren't limited to scary movie soundtracks. "I suspect if we had music that was positive, we would get a similar effect." *Journal reference: PLoS ONE (DOI: 10.1371/journal.pone.0006230)*

Maternal, paternal genes' tug-of-war may last well into childhood

Analysis suggests human development is set by ongoing interplay of parent and offspring genes

CAMBRIDGE, Mass., - An analysis of rare genetic disorders in which children lack some genes from one parent suggests that maternal and paternal genes engage in a subtle tug-of-war well into childhood, and possibly as late as the onset of puberty.

This striking new variety of intra-family conflict, described this week in the Proceedings of the National Academy of Sciences, is the latest wrinkle in the two-decades-old theory known as genomic imprinting, which holds that each parent contributes genes that seek to nudge his or her children's development in a direction most favorable, and least costly, to that parent.

"Compared to other primates, human babies are weaned quite early, yet take a very long time to reach full nutritional independence and sexual maturity," says author David Haig, George Putnam Professor of Organismic and Evolutionary Biology in Harvard University's Faculty of Arts and Sciences. "Human mothers are also unusual among primates in that they often care for more than one child at a time. Evidence from disorders of genomic imprinting suggests that maternal and paternal genes may skirmish over the pace of human development."

Previous research has offered evidence of a genetic struggle for supremacy only during fetal development: In the womb, some genes of paternal origin have been shown to promote increased demands on mothers, leading to fetal overgrowth, while genes of maternal origin tend to have the opposite effect. This new work suggests maternal and paternal genes continue to engage in internal genetic conflict past childbirth.

"This analysis suggests that human life history, and especially humans' unusual extended childhood, may reflect a compromise between what's best for mothers, fathers, and the offspring themselves," Haig says.

Haig delved into clinical case reports on patients with four rare genetic disorders. He found evidence that children with disorders characterized by dominance of some maternal genes -- Silver-Russell syndrome, Prader-Willi syndrome, and Temple syndrome -- place fewer demands on their mothers' resources.

For example, newborns with all three disorders display a weak desire to nurse, and slower childhood growth in general. Many also show early onset of puberty, which often marks a point at which children become less dependent on their mothers' sustenance.

Conversely, babies with Beckwith-Wiedemann syndrome, in which some maternally derived genes are suppressed and paternal genes dominate, are born heavy with particularly large tongues. These individuals usually end up being tall, owing to their rapid growth both in the womb and as young children. They have a high frequency of childhood cancers.

"Clinical data from imprinting disorders suggest paternally-expressed genes promote, and maternally-expressed genes inhibit, childhood growth," Haig writes.

Haig adds that further longitudinal study of feeding and development in individuals with Silver-Russell syndrome, Prader-Willi syndrome, Temple syndrome, and Beckwith-Wiedemann syndrome is needed to more fully understand the role of genomic imprinting in such disorders.

Naming evolution's winners and losers

Mammals, birds show rich species diversity; alligators not so much

Mammals and many species of birds and fish are among evolution's "winners," while crocodiles, alligators and a reptile cousin of snakes known as the tuatara are among the losers, according to new research by UCLA scientists and colleagues. "Our results indicate that mammals are special," said Michael Alfaro, a UCLA assistant professor of ecology and evolutionary biology and lead author of the research.

The study, published July 24 in the early online edition of Proceedings of the National Academy of Sciences, also shows that new species emerge nearly as often as they die off.

Alfaro and his colleagues analyzed DNA sequences and fossils from 47 major vertebrate groups and used a computational approach to calculate whether the "species richness" of each group was exceptionally high or low. The research allows scientists to calculate for the first time which animal lineages have exceptional rates of success.

Among the evolutionary winners are most modern birds, including the songbirds, parrots, doves, eagles, hummingbirds and pigeons; a group that includes most mammals; and a group of fish that includes most of the fish that live on coral reefs, said Alfaro, an evolutionary biologist.

A group with the scientific name Boreoeutheria, which consists of many mammals, has diversified about seven times faster than scientists would have expected, beginning about 110 million years ago, Alfaro and his colleagues calculated. The group includes primates and carnivores, as well as bats and rodents. Pouched mammals, such as kangaroos, are not as richly varied as other mammals, Alfaro said.

Modern birds have diversified about nine times faster than expected, starting about 103 million years ago, and the group of fish that live on coral reefs has diversified about eight times faster than expected, he said.

Who are the evolutionary losers?

Crocodiles and alligators are nearly 250 million years old yet have diversified into only 23 species, Alfaro said. They are diversifying a staggering 1,000 times slower than would have been expected. "Their species richness is so low, given how old they are," he said.

The tuatara, which lives in New Zealand and resembles lizards - although it is actually a distant cousin - has only two species. "In the same period of time that produced more than 8,000 species of snakes and lizards, there were only two species of tuatara," Alfaro said. Why are there not thousands of species of tuataras?

"That is one of the big mysteries about biodiversity," Alfaro said. "Why these evolutionary losers are still around is a very hard thing to explain. They have been drawing inside straights for hundreds of millions of years. It's a real mystery to biologists how there can be any tuataras, given their low rate of speciation. They must have something working for them that has allowed them to persist. In species richness, these are losers, but in another sense, this highlights how unique they are. There are incredibly disparate patterns of species richness."

Tuataras were a bit more diverse in their heyday; there may have been a few dozen species of them, most of which have become extinct, Alfaro said. In contrast, there are more than 9,000 bird species, more than 5,400 mammal species, approximately 5,500 frog species, some 3,000 snake species and 5,200 lizard species, Alfaro said. The number of frog species, although it sounds high, is about what Alfaro would expect, given how old they are - approximately 250 million years old.

"Our analysis suggests we should not be surprised to see a group with that many species in that amount of time," Alfaro said.

There are almost 60,000 species of jawed vertebrates. Alfaro and his colleagues report evidence for exceptional diversification rates in nine taxonomic groups of jawed vertebrates. Interestingly, their findings do not coincide with traditional scientific explanations for why there are so many mammals, birds and fish.

"The timing of the rate increases does not correspond to the appearance of key characteristics that have been invoked to explain the evolutionary success of these groups, such as hair on mammals or mammals' well-coordinated chewing ability or feathers on birds," Alfaro said.

"Our results suggest that something more recent is the cause of the biodiversity. It may be that something more subtle explains the evolutionary success of mammals, birds and fish. We need to look for new explanations."

Co-authors on the PNAS paper are Luke Harmon, a professor of biological sciences at the University of Idaho; Francesco Santini, a UCLA postdoctoral scholar in Alfaro's laboratory; Chad Brock, a graduate student of biology at Washington State University; Hugo Alamillo, a graduate student of biology at Washington State

University; Alex Dornburg, a former undergraduate in Alfaro's laboratory, now a graduate student at Yale University; Daniel Rabosky, a graduate student of biology at Cornell University; and Giorgio Carnevale, a postdoctoral scholar at Italy's University of Pisa.

The research is federally funded by the National Science Foundation.

Alfaro's laboratory also studies why some groups of animals have great diversity in their shapes and others do not, even if there are many species. He and his colleagues use DNA sequencing to tease apart evolutionary relationships, analyze the fossil record and conduct sophisticated statistical analysis.

"We are interested in understanding the causes of biodiversity," Alfaro said. "We are trying to understand what explains the staggering diversity of reef fishes and other vertebrates."

"Our analysis can highlight how much higher extinction rates are in the present, compared with the historical rates," he said. *For more about Alfaro's research, visit his website at http://alfarolab.eeb.ucla.edu.*

US guns fuel Canada and Mexico crimes, UK gun crime remains rare

Special issue of Criminology and Criminal Justice explores gun crime internationally

Los Angeles, London, New Delhi, Singapore and Washington DC (29 July 2009) – Guns smuggled from the US arm criminals in Canada and Mexico, contributing to a higher murder rate in Canada and more intense drug crime conflict near the Mexican border, according to a study published today in a special issue of Criminology and Criminal Justice, published by SAGE.

However, authors Philip J. Cook, of Duke University Durham, NC, US, Wendy Cukier Ryerson of the University of Toronto, Canada and Keith Krause from the Graduate Institute of International and Development Studies Geneva, Switzerland highlight a dearth of empirical evidence on gun crime available to criminologists. Gun violence in North America remains the subject of considerable speculation and debate. In their paper The Illicit Firearms Trade in North America, the authors draw upon economics concepts, examining gun crime in the context of each country's regulatory framework.

The US is undoubtedly a major supplier of illegal guns (particularly handguns) to both Canada and Mexico. But limited data hamper efforts to predict the effect of a successful crackdown on illegal firearms by US authorities, the authors suggest. Both policy makers and law enforcement would benefit from research to fill these information gaps.

The data that are available show that the majority of traced handguns recovered from Canadian crime scenes originate in US. Another major source of illegal guns in Canada, and in many other countries is "leakage" from state stockpiles (police and military) through theft, corruption or other means. For instance, 'insiders' illegally sold over 3000 firearms recovered in crime or surrendered in amnesties to the Metropolitan Toronto Police Service.

Investigators have traced 90 to 95 percent of weapons in Mexico to the US, but how did they get there? The guns sampled may not represent the bigger picture: the figure reflects firearms submitted for tracing by Mexican authorities. Authorities recover only a fraction of firearms from crimes and gun battles, and traces are only requested on some recovered weapons.

Central America, a region awash with weapons imported by both governments and rebel groups during the civil wars in El Salvador, Nicaragua and Guatemala, is a further potential weapon source to Mexico, as are Chinese, Russian, Eastern European, or other sources. To date evidence is mainly anecdotal. Still less is known about the third source of weapons, the Mexican security forces themselves. The Small Arms Survey 2008 showed that weapons diverted from police and armed forces are a major and sometimes the main source of illicit weapons in many countries.

Some weapons used in Mexican crimes such as grenades, RPGs and fully automatic weapons are less easy to acquire in the US, and have probably arrived from elsewhere. This contrasts with Canada, where very few cases detail handguns from anywhere but the US, other than arms illegally diverted from legal Canadian supplies.

According to Cook, the specific impact and effects of illicitly trafficked firearms are unknowns. "Although we know that armed violence can have a variety of deleterious effects on perceived and real insecurity, public health, economic development, and political stability, we do not know how much of this can be associated specifically with changes in the availability of firearms," he says.

Some values can be quantified: Previous research has shown that life expectancy is lowered by 0.6 years for all Mexicans as a result of armed violence, with the US and Canada figures at 0.31 and 0.08, respectively. But firearms' negative effects are highly context dependent, with factors such as demand strength, types of weapons circulating, social groups with weapons access and reasons they possess them all contributing to the mix.

"The use of guns by criminal groups increases their relative power, and in the dramatic circumstances we see in Mexico, contributes to subverting legitimate authority and creating such fear as to have a substantial economic and political impact," says Cook.

The rate of gun homicide in Canada is statistically low and falling, yet public perception is that gun crime is rising. When Toronto, a city with 2.8 million people hit 52 gun homicides in 2005, it became "the year of the gun" in spite of the fact that the city had one of the lowest murder rates on the continent for a city of its size. Rates of homicide with guns are 6.7 times higher in the US than in Canada, and the US has 5.1 times Canada's rate per 100,000 of gun robberies.

The authors speculate US authorities would not only have to stem the supply of smuggled weapons from the US, but also other potential sources to successfully block the flow of deadly arms to criminals and criminal organizations.

Statements made by public officials are usually intended to influence public opinion by offering conclusions, rather than to inform researchers' analyses, the authors believe. They call for more data from criminal investigations and gun tracing to be made available to researchers.

"A broader inquiry is warranted," says Cook. "The stakes are very high for developing effective strategies for limiting the illicit movements of guns."

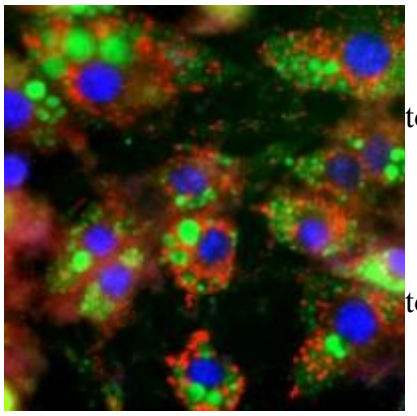
Another paper in the same issue on firearms discusses the UK and the Netherlands, which have among the lowest occurrence of gun-homicide in advanced industrial democracies. In Third Wave Criminology, guns, crime and social order, Adam Edwards of Cardiff University, UK and James Sheptycki, of York University, Canada use these examples to illustrate the evolution of criminology in the context of evolving paradigms from the sociology of science in the wake of postmodernism, and towards a basis for action in the face of scientific uncertainty.

Criminology and Criminal Justice Volume 9 Number 3 August 2009 is published today (29th July 2009) by SAGE. All articles in the special issue will be freely available for a limited time from http://crj.sagepub.com/. For interviews with authors from the special issue contact Mithu Mukherjee, mithu.mukherjee@sagepub.co.uk/+44(0)20 7324 2223.

Scientists create energy-burning brown fat in mice

Suggests novel strategy for treating obesity in people

Boston--Researchers at Dana-Farber Cancer Institute have shown that they can engineer mouse and human cells to produce brown fat, a natural energy-burning type of fat that counteracts obesity. If such a strategy can be developed for use in people, the scientists say, it could open a novel approach to treating obesity and diabetes.



A team led by Bruce Spiegelman, PhD, has identified both parts of a molecular switch that normally causes some immature muscle cells in the embryo to become brown fat cells. With this switch in hand, the scientists showed they could manipulate it to force other types of cells in the laboratory to produce brown fat, known as Brown Adipose Tissue (BAT). Their findings are being reported in the journal Nature on its Web site as an advanced online publication on July 29.

This is a microscope image of brown fat (e-BAT, or engineered Brown Adipose Tissue) created by adding a key control switch to skin cells of mice. Presence of green-stained objects (droplets of oil stored in the cell) confirms the skin cells have been converted to brown fat-producing cells. Blue objects are cell nuclei. **Shingo Kajimura, Ph.D., Dana-Farber Cancer Institute**

The scientists then transplanted these synthetic brown fat precursors, known as eBAT (engineered BAT), into adult mice to augment their innate stores of brown fat. Tests showed that the brown fat transplants were burning caloric energy at a high rate -- energy that otherwise would have been stored as fat in white adipose tissue.

"Since brown fat cells have very high capacity to dissipate excess energy and counteract obesity, eBAT has a very high potential for treating obesity," said Shingo Kajimura, PhD, lead author of the paper. "We are currently working on this."

Excess caloric energy in the diet is stored in white fat calls that pile up in the body, particularly in the thighs and abdomen. The accumulated fat content in overweight people puts stress on these cells, which give out signals that cause inflammation in body organs and the circulatory system, creating risks of heart disease and diabetes.

Brown fat, by contrast, works in an opposite fashion; it evolved to protect animals from cold conditions and prevent obesity. Brown fat cells are equipped with a large supply of mitochondria -- tiny organelles that use oxygen to burn sugar from the diet to generate heat, rather than store the energy as fat.

Scientists have long thought that brown fat was present in young animals and human newborns but virtually absent in human adults. Recently, however, researchers have used modern PET (positron emission tomography) scanners -- which detect tissue that is actively absorbing sugar -- to search for deposits of brown fat in adults.

Such experiments have revealed unexpectedly large amounts of brown fat scattered through the neck and chest areas.

In 2007, Spiegelman's team, led by Patrick Seale, PhD, who is the second author of the new Nature paper, discovered a protein, PRDM16, that serves as a switch that determines whether immature muscle cells will develop into mature muscle cells or become brown fat cells.

But this was not the whole story. The scientists suspected that PRDM16 worked with another unknown protein to initiate brown fat development. This proved to be the case. In the new experiments, the Spiegelman group found that PRMD16 works in tandem with the protein C/EBP-beta, and only as a two-part unit are they sufficient to jump-start brown fat development in several types of cells.

To find out if the PRDM16-C/EBP-beta switch could change the identity of other types of cells, forcing them to become brown fat cells, the researchers used viruses to transfer the switch into embryonic mouse connective tissue cells called fibroblasts. They also installed the switch into adult mouse skin cells, and into human skin cells isolated from foreskins removed from newborns during circumcision.

In all three cases, the fibroblasts produced mature brown fat cells. The scientists then transplanted the cells into mice, where they produced brown fat tissue. PET scans confirmed that the new brown fat tissue was burning excess energy in the animals, as they should. The experiments did not test whether the extra brown fat actually protected the mice from becoming obese.

Spiegelman said the results "give a lot more credence" to efforts to manipulate the brown fat switch as a potential means of treating people with obesity and diabetes. One strategy would be to remove some tissue from the patient, add the PRDM16-C/EBP switch, and return it to the patient where it would manufacture additional brown fat.

A more conventional possibility, Spiegelman said, would be to administer a drug to the patient that would ramp up the production of brown fat without the need for a transplant. "If we can find a hormone that does that, it's reasonable to think that it might provide a direct anti-obesity treatment."

Other authors on the paper are Kazuishi Kubota, PhD, and Steven P. Gygi, PhD, of Harvard Medical School, and Elaine Lunsford and John V. Frangioni, MD, PhD, of Beth Israel Deaconess Medical Center.

The research was supported by grants from the National Institutes of Health and the Picower Foundation.

Bizarre walking bat has ancient heritage

A bizarre New Zealand bat that is as much at home walking four-legged on the ground as winging through the air had an Australian ancestor 20 million years ago with the same rare ability, a new study has found.

The discovery overturns a long-held held view that the agile walking and climbing skills of the lesser short-tailed bat - Mystacina tuberculata – evolved in the absence of any ground-dwelling mammal competitors or predators, says an international team of researchers led by Dr Suzanne Hand, a bat expert at the University of New South Wales, Sydney.

Along with the American common vampire bat - Desmodus rotundus - the NZ bat is one of only two of 1,100 bat species worldwide that has a true four-legged walking gait when manoeuvring on the ground.

It uses its wings as forelegs. Its thumb and toe claws have a unique extra talon for extra grip, plus a system of adhesive, gecko-like grooves in the soft, deeply wrinkled soles of its feet,

The team has found that other special muscle and bone adaptations were also present in one of its extinct rainforest-dwelling Australian ancestors, fossils of which have been found at the rich Riversleigh World Heritage Fossil Site in north-west Queensland, it says in a report published in the journal BMC Evolutionary Biology.

"The lesser short-tailed bat seems to be the sole survivor of an ancient Australian lineage now found only in New Zealand," says Dr Hand. "This study shows that, contrary to existing hypotheses, bats are not overwhelmingly absent from the ground because of competition from, or predation by, other mammals.

"Unlike for birds, there is currently no evidence that any bat has evolved a reduced capacity for flight as a result of isolation on islands.

"Rather, it would seem that walking is rare in bats because it has advantages for them only in special circumstances. Competition with other mammals and pressure from terrestrial predators does not deter modern vampire bats from walking. Likewise, the rich rainforest environment in which the ancestors of the mystacinid bats evolved in ancient Australia was teeming with ground-based competitors and predators."

A small secretive creature with velvety fur, the lesser short-tailed bat is New Zealand's only terrestrial mammal: it spends long periods on the ground in heavily forested areas, hunting insects and seeking out fruit, nectar and pollen.

It also appears to have evolved a special relationship as a pollinator for the Hades flower, or woodrose, a parasitic plant that produces nectar from blooms near ground level at the base of tree trunks.

Among other unusual traits, the bat is thought to use its teeth and claws to excavate roosting burrows and males appear to compete for mates by gathering for singing competitions in their breeding season.

Video of the walking bat can be seen here:
http://www.arkive.org/lesser-short-tailed-bat/mystacina-tuberculata/video-00.html
The online paper can be found here: http://www.biomedcentral.com/1471-2148/9/169
An online digital skeleton of Mystacina tuberculata can be found here:
http://digimorph.org/specimens/Mystacina_tuberculata/whole/

Freshly crushed garlic better for the heart than processed

A new study reports what scientists term the first scientific evidence that freshly crushed garlic has more potent heart-healthy effects than dried garlic. Scheduled for the Aug. 12 issue of the Journal of Agricultural and Food Chemistry, it also challenges the widespread belief that most of garlic's benefits are due to its rich array of antioxidants. Instead, garlic's heart-healthy effects seem to result mainly from hydrogen sulfide, a chemical signaling substance that forms after garlic is cut or crushed and relaxes blood vessels when eaten.

In the study, Dipak K. Das and colleagues point out that raw, crushed garlic generates hydrogen sulfide through a chemical reaction. Although best known as the stuff that gives rotten eggs their distinctive odor, hydrogen sulfide also acts as a chemical messenger in the body, relaxing blood vessels and allowing more blood to pass through. Processed and cooked garlic, however, loses its ability to generate hydrogen sulfide.

The scientists gave freshly crushed garlic and processed garlic to two groups of lab rats, and then studied how well the animals' hearts recovered from simulated heart attacks. "Both crushed and processed garlic reduced damage from lack of oxygen, but the fresh garlic group had a significantly greater effect on restoring good blood flow in the aorta and increased pressure in the left ventricle of the heart," Das said.

Article #2 For Immediate Release "Freshly crushed garlic is a superior cardioprotective agent than processed garlic"
Download Full Text Article: http://pubs.acs.org/stoken/presspac/presspac/full/10.1021/jf901301w

Research shows rates of severe childhood obesity have tripled

Winston-Salem – Rates of severe childhood obesity have tripled in the last 25 years, putting many children at risk for diabetes and heart disease, according to a report in Academic Pediatrics by an obesity expert at Brenner Children's Hospital, part of Wake Forest University Baptist Medical Center.

"Children are not only becoming obese, but becoming severely obese, which impacts their overall health," said Joseph Skelton, M.D., lead author and director of the Brenner FIT (Families in Training) Program. "These findings reinforce the fact that medically-based programs to treat obesity are needed throughout the United States and insurance companies should be encouraged to cover this care."

The research was published on-line and will appear in the September print edition. Skelton and colleagues compared data from the National Health and Nutrition Examination Survey (NHANES). They looked at the prevalence of obesity and severe obesity in a study population of 12,384 children, representing approximately 71 million U.S. children ages 2 to 19 years.

Severe childhood obesity is a new classification for children and describes those with a body mass index (BMI) that is equal to or greater than the 99th percentile for age and gender. For example, a 10-year-old child with a BMI of 24 would be considered severely obese, Skelton said, whereas in an adult, that is considered a normal BMI. An expert committee convened by the American Medical Association, the Centers for Disease Control and the Department of Health and Human Services proposed the new classification in 2007.

The research by Skelton and colleagues is the first of its kind to use the new classification and detail the severity of the problem. They found that the prevalence of severe obesity tripled (from 0.8 percent to 3.8 percent) in the period from 1976-80 to 1999-2004. Based on the data, there are 2.7 million children in the U.S. who are considered severely obese.

Increases in severe obesity were highest among blacks and Mexican-Americans and among those below the poverty level. For example, the percentage of Mexican-American children in the severely obese category was 0.9 percent in 1976-80 and 5.2 percent in 1999-2004.

Researchers also looked at the impact of severe obesity and found that a third of children in the severely obese category were classified as having metabolic syndrome, a group of risk factors for heart attack, stroke and diabetes. These risk factors include higher-than normal blood pressure, cholesterol and insulin levels.

"These findings demonstrate the significant health risks facing this morbidly obese group," wrote the researchers in their report. "This places demands on health care and community services, especially because the highest rates are among children who are frequently underserved by the health care system."

The research was supported, in part, by the National Institutes of the Health and the Robert Wood Johnson Foundation. Co-researchers were: Stephen Cook, M.D., M.P.H.; Peggy Auinger, M.S.; Jonathan Klein, M.D., M.P.H.; University of Rochester School of Medicine and Dentistry; and Sarah Barlow, M.D., M.P.H., Baylor College of Medicine.

Enigma of the 23-year-old baby

* 29 July 2009 **by Holly Tucker**

The "cabinets of curiosity" of the 16th and 17th centuries housed the extraordinary souvenirs that European missionaries and other travellers brought back from the New World and the East. Stuffed birds with brilliantly coloured plumage sat alongside seashells larger than the human body and mummies plucked from Africa's desert sands. But, as French surgeon Pierre Dionis discovered, sometimes the marvels in your own backyard are the strangest of all. When Dionis stumbled across a leathery fetus-like object in a priest's collection, he resolved to learn the truth about it. Could it really be that this misshapen object was the product of a 23-year-long pregnancy?

IN 1678, Pierre Dionis, surgeon in the court of Queen Maria Theresa, accompanied his illustrious patient on a pilgrimage to Pont à Mousson, a small hamlet in north-east France. There, the royal entourage was greeted by the village priest, Father Babilart, a man with a reputation for collecting strange trinkets from around the globe. Babilart was eager to show off his playthings and after ducking skeletons that dangled from the ceiling and side-stepping columns pillaged from Roman ruins, he emerged with a prize he was sure would please both the queen and her companion.

The priest lifted a large jar filled with distilled spirits into the air. Floating inside was a leathery ball-shaped object. Intrigued, the queen and Dionis moved in closer to get a better look. The ball, they now saw, was a horrifically deformed fetus. "Its arms, legs, and spine were so crumpled up together it was impossible to stretch them," Dionis wrote. "Its face was hideous, and was ruddy reddish brown in colour." The priest explained proudly that the child had been cut from its mother's belly after her death - and 23 years after she learned she was pregnant.

The queen left Dionis to investigate the priest's claims. Maria Theresa's fascination for anatomy was well known. Her husband, Louis XIV, kept a menagerie of exotic animals, and whenever one died she summoned the surgeon to dissect it. "The Queen did not have the same repugnance that other women have for anatomical demonstration," Dionis later explained in his influential Dissertation on the Generation of Man in 1698.

The queen's surgeon was no stranger to medical mysteries. At Versailles, he often performed autopsies to determine the cause of death when noblemen succumbed to illness, accident or foul play. Nor was he a stranger to "generation", as reproduction and childbirth were called at the time. The king had appointed Dionis to teach anatomy in the royal gardens, and there he had dissected pregnant women whose corpses had been cut from the gallows or, in the case of courtly women, offered for study by the royal family.

Dionis questioned Babilart at length, but the normally effusive man "could not - or did not want - to tell me more". Undeterred, Dionis pressed on. Decades-long pregnancies were not unheard of in the Renaissance and the stories continued into the 17th century. In fact, such tales of interminable childbearing were regularly told and retold in this era before caesarean section and ultrasound.

Dionis was familiar with the story of the famous "Fetus of Sens". In 1582, 68-year-old Colombe Chatri had died following 28 years of pregnancy. When doctors dissected her body, they found a monstrous, stone-like child - a girl, as it turned out. A more recent case had surfaced in 1678 in Toulouse, where 64-year-old Marguerite Mathieu had died with a 25-year-old fetus still in her womb.

The biological explanation behind these earlier cases had not been determined with any certainty, but Dionis paid no heed to the old wives' tales that blamed magic and witchcraft. Although witch trials were on the wane in Europe by this time, expectant mothers were still warned about midwives who dabbled in the dark arts. Privy to knowledge that could help - or hinder - childbirth, midwives were both necessary and feared. In the Mathieu case, the general belief was that the mother had turned down a midwife's services. In return, the angry midwife cast a spell on the child.

Dionis steeled himself for such explanations as he set off to interview the villagers. Instead, what he heard was surprisingly to the point. The mother of the fetus in question had indeed been pregnant for 23 years, after producing several other children without incident. She had died from illness, not from the pregnancy. After her death in 1674, surgeons had been called in to perform an autopsy. They discovered the fetus that was now in Father Babilart's safe-keeping.

The surgeon reviewed what he knew about reproductive anatomy and physiology. The two great medical philosophers of antiquity, Hippocrates and Galen, had held that conception was the result of a propitious mixing of male and female "seeds" during intercourse. The invention of the microscope had led to the discovery of human eggs in 1672, and of spermatozoa five years later. This meant that centuries-old theories on reproduction needed to be rewritten.

Two schools of thought quickly emerged: reproduction was deemed the sole responsibility of either the female egg or the male sperm - but not both. "Ovists" held that all of humanity lay preformed in women's eggs, nestled one inside the other like Russian dolls; "animaculists" argued that preformed little men lay in wait, each in the head of a sperm. For nearly a century, theories that humans were preformed in either the egg or sperm dominated embryological theory. It was not until the 1780s that early ideas about what we might now recognise as epigenesis would begin to take hold in medicine.

Dionis was an ovist but was nevertheless appalled by suggestions that sexual intercourse might not be necessary for conception. In the south of France, an "absurd rumour" had been circulating about a young man who became pregnant after failing to ejaculate during sex. A few hours later, the man felt excruciating pain in one of his testicles. Over the next eight or nine months, the testicle had swollen to the size of a turkey's egg and required amputation. Inside the testicular mass, doctors found "a bony globe that had two orbits with two small black pouches, which were full of water". While some claimed it was possible that the man had impregnated himself, Dionis held firm in his conclusion that this testicular "pregnancy" was simply a cancerous mass. Determined to solve the mystery of the 23-year-old fetus, Dionis returned to Babilart's cabinet of curiosity and pressed the priest for more information. Babilart reluctantly offered his own explanation for the interminable pregnancy. The woman had been pregnant not for 23 years but for her entire life, he said. An egg had lodged in her abdomen while she was still in her own mother's womb. The fetus was not her child, but rather, her twin.

Dionis dismissed the priest's speculations: in his opinion the man was as deluded as those who spoke about testicular pregnancies. The surgeon explained to Babilart with growing impatience that "seminal vapour" released during the sex act had moved up through the woman's uterus and into the fallopian tubes. The seminal vapour had caused the preformed, miniature fetus in one of the tubes to expand and grow. But instead of travelling along the tube and into the uterus, where it would develop over the term of the pregnancy, the fetus "dropped into the stomach", where it had remained until the mother's death. The priest "refused to yield to my reason", Dionis later wrote. "So I decided to leave him alone in his stubbornness."

Ignoring his preformationist leanings, Dionis's theory follows the general lines of what we would now recognise as an ectopic or abdominal pregnancy. But was the priest necessarily wrong? His explanation is suggestive of fetus in fetu (fetus within in a fetus), sometimes known as a "parasitic twin". First recognised in the 19th century, this is a rare condition in which a calcified, fetus-like mass lodges in the body of another while both are in the womb. Perhaps the old priest's claim was not so improbable after all.

UCSF researchers identify new drug target for Kaposi's Sarcoma

UCSF researchers have identified a new potential drug target for the herpes virus that causes Kaposi's sarcoma, re-opening the possibility of using the class of drugs called protease inhibitors against the full herpes family of viruses, which for 20 years has been deemed too difficult to attain.

The new drug target, which is known as a protease dimer, could serve as a model for developing new therapeutics for diseases ranging from cancer to Alzheimer's, the researchers say. Findings are reported in the Advance Online Publication section of the "Nature Chemical Biology" web site and can be found at http://www.nature.com/nchembio/index.html .

Most current antiviral drugs target the active sites of viral proteins, where enzymes and receptors work in a lock-and-key approach to either activate or deactivate that particular protein, the researchers explained. Traditionally, drug development has focused on inhibiting that lock-and-key action to prevent the enzyme, or receptor from being effective.

Some viral enzymes known as proteases, however, including those for HIV and the herpes virus family, take the form of a dimer, or two identical halves – much like a fully opened clamshell – in their most stable state. Those proteases play an essential role in making the virus infectious, but require the two clamshell halves to bind together to be activated, according to the paper.

The HIV protease was successfully targeted for drug development in the 1980s, by blocking the active site on the surface of the dimer, but the herpes virus protease dimer has consistently eluded efforts to disrupt it at its active site, the researchers said.

The UCSF team set out to find ways to instead prevent the two halves of the dimer from connecting at that clamshell joint, to prevent it from activating. What they found was a new target on the unstable, monomer form of the protease, which responded well to a chemical inhibitor.

“If you disrupt the protein-protein interactions, you don’t need the key to a specific lock,” said Charles S. Craik, PhD, senior author on the paper and a professor of pharmaceutical chemistry in the UCSF School of Pharmacy. “Instead, we’re essentially preventing the lock from being made in the first place.”

Craik, who also led a team that identified HIV protease inhibitors in the late 1980s, said the “Nature Chemical Biology” paper validates this new site as a viable option for small-molecule drugs to treat Kaposi’s, as well as other members of this viral family.

“All known herpes virus proteases are structurally similar,” Craik explained. “The inhibitor we found knocks out not only KS, but also the cytomegalovirus protease, so the site we’ve identified here could be a target for a broad-acting inhibitor against the entire viral family.”

To their knowledge, the researchers said, this is the first small-molecule inhibitor of a herpes virus protease to not only act outside the active site, but also to select for the partially unfolded protein to keep it from forming the dimer interface.

Herpes viruses make up one of the most prevalent viral families, including eight human viruses that cause a variety of devastating illnesses, the researchers said. Those include mononucleosis (Epstein-Barr virus), shingles (Varicella zoster virus), genital herpes (herpes simplex), retinitis (cytomegalovirus) and cancer (Kaposi’s sarcoma). While therapies exist for these viruses, they often have negative side effects and are facing rising viral resistance.

In addition to validating herpes virus proteases as suitable targets, Craik said this research was also among the first to use computational design to identify and create a potential drug to target that protease interface.

Using high-throughput screening, the team screened a library of 182 compounds that it had specifically and rationally designed to mimic the protease interface. The work identified six molecules that inhibited the Kaposi’s sarcoma virus protease activity by at least 50 percent, including one that was highly potent.

That discovery potentially opens myriad opportunities for drug discovery, Craik said, by making target receptors that were biologically validated, but then deemed undruggable, more attractive. Protein-protein interactions have been researched as drug targets against a range of diseases, from certain cancers to neurodegenerative diseases. This advance could enable researchers to reconsider those targets, he said.

The lead investigator on the paper was Tina Shahian, with the Graduate Group in Biochemistry and Molecular Biology at UCSF. Co-authors were Gregory M. Lee and Ana Lazic, both in the UCSF Department of Pharmaceutical Chemistry; and Leggy A. Arnold, Priya Velusamy, Christina M. Roels and R. Kiplin Guy, all with the Department of Chemical Biology and Therapeutics at St. Jude Children’s Research Hospital, Memphis, TN.

The CMV protease expression plasmid for this work was provided by Wade Gibson, a professor in the Department of Pharmacology and Molecular Sciences at Johns Hopkins School of Medicine. The work was funded by grants from the National Institutes of Health, the American Lebanese and Syrian Associated Charities and St. Jude Children’s Research Hospital. The authors declare no conflict of interest in this paper.

Did an ice age boost human brain size?

* 29 July 2009 **by Bob Holmes**

IT IS one of the biggest mysteries in human evolution. Why did we humans evolve such big brains, making us the unrivalled rulers of the world?

Some 2.5 million years ago, our ancestors' brains expanded from a mere 600 cubic centimetres to about a litre. Two new studies suggest it is no fluke that this brain boom coincided with the onset of an ice age. Cooler heads, it seems, allowed ancient human brains to let off steam and grow.

For all its advantages, the modern human brain is a huge energy glutton, accounting for nearly half of our resting metabolic rate. About a decade ago, biologists David Schwartzman and George Middendorf of Howard University in Washington DC hypothesised that our modern brain could not have evolved until the Quaternary ice age started, about 2.5 million years ago. They reckoned such a large brain would have generated heat faster than it could dissipate it in the warmer climate of earlier times, but they lacked evidence to back their hypothesis.

Now hints of that evidence are beginning to emerge. Climate researcher Axel Kleidon of the Max Planck Institute for Biogeochemistry in Jena, Germany, modelled present-day temperature, humidity and wind conditions around the world using an Earth-systems computer model. He used these factors to predict the maximum rate at which a modern human brain can lose heat in different regions. He found that, even today, the ability to dissipate heat should restrict the activity of people in many tropical regions (Climatic Change, vol 95, p 405).

If keeping cool is a problem now, Kleidon says, it would have been even more challenging - perhaps too challenging - 2 or 3 million years ago when temperatures were a few degrees warmer than today and air-conditioning units were harder to come by.

A new study by Schwartzman and Middendorf suggests that a small drop in global temperatures may have made a big difference. The pair used basic equations of heat loss to estimate how fast the small-brained Homo habilis would have been able to cool off. Assuming overheating limited the size of H. habilis's brain, they then calculated what drop in air temperature would have been needed for Homo erectus to be able to support its bigger brain (see diagram). They found that a drop in air temperature of just 1.5 °C would have done the trick (Climatic Change, vol 95, p 439).

Given the timescales involved, it may be near-impossible to match definitively the onset of an ice age with speciation, but a 1.5 °C drop is consistent with the cooling climate of the time, says Middendorf.

"In principle, I'm receptive to the hypothesis," says Dean Falk, a palaeoanthropologist at Florida State University in Tallahassee, "but I need the data." She says that if measurements showed that people living in tropical countries today have smaller brains relative to their body size than people in temperate climates, this would go against expectation and lend support to Kleidon's model.

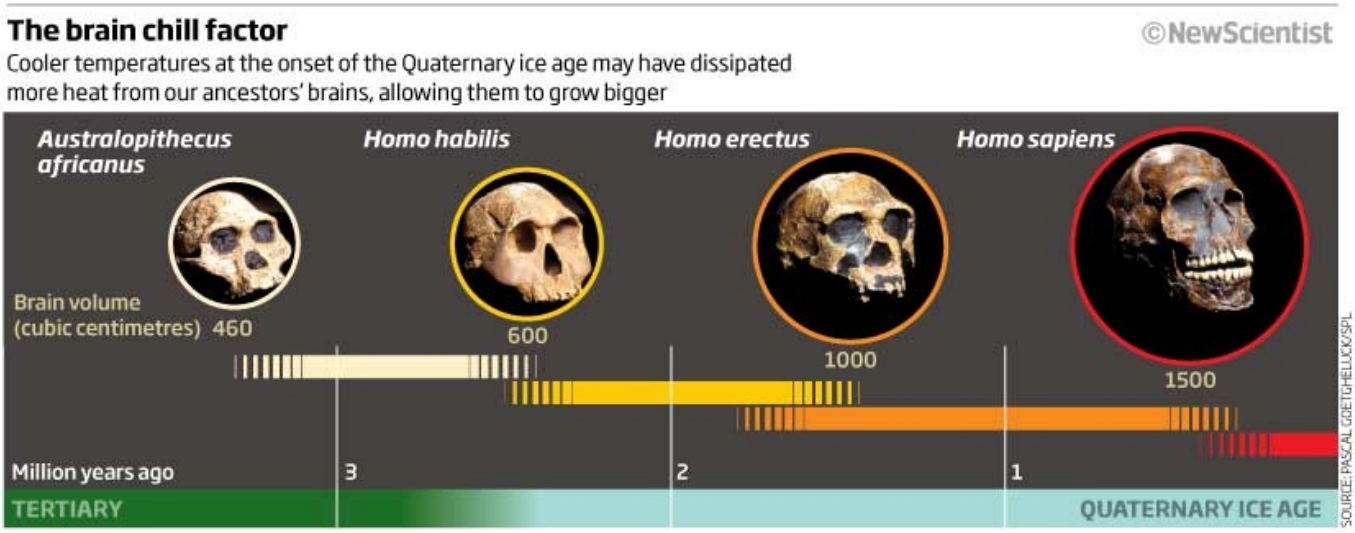
Being able to cool bigger brains can only be part of the story, however. It would have lifted the brakes on expansion, says psychologist David Geary at the University of Missouri in Columbia, but there has to be something driving the increase.

Over the years, researchers have come up with three broad reasons why bigger brains might have been advantageous: to give their owners the ability to cope with changing climates by exploiting technologies such as shelter, fire and clothing; to deal with the cognitive demands of hunting and gathering; or to help people outsmart their neighbours.

To help narrow this down, Geary collected data from 175 fossil hominin skulls, from 1.9 million to 10,000 years old. Then he looked to see whether brain size was best correlated with climatic variability - a crude measure of biodiversity which could indicate the complexity of hunting and gathering - or the human population size at the time, which could reflect the complexity of social interactions.

Geary's analysis found that population size was the best predictor of brain size, suggesting that our ancestors' need to outcompete their neighbours in order to survive may have been the strongest driver of brain growth (Human Nature, vol 20, p 67).

The case is far from closed - Geary's study does not demonstrate cause and effect, for one thing - but the picture beginning to emerge suggests that an ice age set the stage for a socially driven brain boom. And from that time on, it was the brainiacs who stole the show.



Greenhouse brains

If global cooling allowed humans to evolve their big brains, will today's global warming take them away again? "I'd hate to think that a difference of 1.5 °C might mean the end of humans because our brains cook," says George Middendorf of Howard University in Washington DC, "but I guess it's a scenario that might play out."

It probably won't, though, thanks to what those big human brains made possible: culture.

"When culture comes in, it layers itself on top of the biological constraints," says Tyler Volk, an Earth-systems expert at New York University. Thanks to culture and technology, we now have ways of buffering ourselves against hot climates, not only with air conditioning, but also with basic tools such as fans, thick-walled buildings and reservoirs to ensure we have plenty of water.

Only one thing could destroy that buffer - a total breakdown of society.

Chimps born to appreciate music

Chimpanzees are biologically programmed to appreciate pleasant music. The discovery comes from experiments showing that an infant chimpanzee prefers to listen to consonant music over dissonant music. That suggests the apes are born with an innate appreciation of pleasant sounds, say scientists in the journal Primates.

Until now, this was thought to be a universal human trait, but the new finding suggests it evolved in the ancestors of humans and modern apes.

Tasuku Sugimoto and Kazuhide Hashiya of Kyushu University in Hakozaki and colleagues in Japan tested how a young captive chimpanzee named Sakura responded to music as she aged from 17 weeks to 23 weeks old.

Sakura had been abandoned by her mother, forcing members of the staff at Itozu-no-mori Park in Fukuoka where she lived to care for her. Crucially, she had never been exposed to any form of music before she took part in the trials.

During the experiments, Sakura lay on a bed while a woollen string was attached to her right hand, allowing the infant chimp to pull on the cord at will. A music player and speakers was then set up around her, playing melodies lasting between 38 and 63 seconds long. Every time Sakura pulled on the cord, the music would be repeated. During six trials, conducted one a week for six weeks with each lasting around 20 minutes, the researchers played Sakura a range of tunes.

One was a 38 second minuet from Duette Englischer Meister in F major. Another, a 38 second minuet from a handwritten sheet of German music composed in 1720.

These consonant tunes were also adjusted using orchestration software to make them dissonant. For example, all the Gs in the 38 second Duette Englischer Meister music were altered to G-flat and all the Cs to C-flat, creating 32 dissonant intervals.

In three of the six trials, the researchers first played Sakura the more pleasant consonant music and in the others, they started with the less pleasant sounding dissonant music.

Play it again

Across all six sessions, Sakura pulled on the cord to voluntarily listen to the pleasurable music significantly more often than to the dissonant passages.

"Our main surprise was the results being so consistent," says Hashiya. "She rapidly learnt the rule of the setup and consistently produced consonant music over dissonant music for longer duration."

The discovery that an infant chimp, with no prior exposure to music, innately prefers to listen to consonant melodies could have important implications for how an appreciation for music evolved.

"Music is one of the universal human natures beyond cultures, just like language," says Hashiya.

But it was always thought that it was a uniquely human trait, one present even in babies just a few days old.

"The preference for consonant music over dissonant music in an infant chimpanzee has implications for the debate surrounding human uniqueness in the capacity for music appreciation," the researchers write in Primates.

Experiments have shown that various bird species can differentiate between consonant and dissonant sounds, but they do not actively prefer listening to one over the other. Other research on cotton-top tamarin monkeys also found no such preference.

But Sakura's appreciation for consonant melodies "specifically suggests that one of the major factors that constitute musical appreciation might not be unique to humans: instead it might be something that we share with our phylogenetically closest relatives," say the researchers.

Hashiya explains that it is very difficult to rule out whether young human infants have had prior exposure to music on the radio or in their family's house before they are tested.

"To figure out the response of Sakura, we have to consider her lack of music experience, which should draw a clear contrast with ordinary human infants. It supports the view that the preference is independent of cultural experience," he says.

The researchers hope to study the effect further. For now they speculate that the chimps' innate preference for pleasurable melodies may serve some biological function in the wild, perhaps helping them detect other chimps' voices above other sounds, for example.

Crashing comets not likely the cause of Earth's mass extinctions

Scientists have debated how many mass extinction events in Earth's history were triggered by a space body crashing into the planet's surface. Most agree that an asteroid collision 65 million years ago brought an end to the age of dinosaurs, but there is uncertainty about how many other extinctions might have resulted from asteroid or comet collisions with Earth.

In fact, astronomers know the inner solar system has been protected at least to some degree by Saturn and Jupiter, whose gravitational fields can eject comets into interstellar space or sometimes send them crashing into the giant planets. That point was reinforced last week (July 20) when a huge scar appeared on Jupiter's surface, likely evidence of a comet impact.

New University of Washington research indicates it is highly unlikely that comets have caused any mass extinctions or have been responsible for more than one minor extinction event. The work also shows that many long-period comets that end up in Earth-crossing orbits likely originate from a region astronomers have long believed could not produce observable comets. A long-period comet takes from 200 years to tens of millions of years to make a single orbit of the sun.

"It was thought the long-period comets we see just tell us about the outer Oort Cloud, but they really give us a murky picture of the entire Oort Cloud," said Nathan Kaib, a University of Washington doctoral student in astronomy and lead author of a paper on the work being published July 30 in Science Express, the online edition of the journal Science.

The Oort Cloud is a remnant of the nebula from which the solar system formed 4.5 billion years ago. It begins about 93 billion miles from the sun (1,000 times Earth's distance from the sun) and stretches to about three light years away (a light year is about 5.9 trillion miles). The Oort Cloud could contain billions of comets, most so small and distant as to never be observed.

There are about 3,200 known long-period comets. Among the best-remembered is Hale-Bopp, which was easily visible to the naked eye for much of 1996 and 1997 and was one of the brightest comets of the 20th century. By comparison, Halley's comet, which reappears about every 75 years, is perhaps the best-known comet, but it is a short-period comet, most of which are believed to originate in a different part of the solar system called the Kuiper Belt.

It has been believed that nearly all long-period comets that move inside Jupiter to Earth-crossing trajectories originated in the outer Oort Cloud. Their orbits can change when they are nudged by the gravity of a neighboring star as it passes close to the solar system, and it was thought such encounters only affect very distant outer Oort Cloud bodies.

It also was believed that inner Oort Cloud bodies could reach Earth-crossing orbits only during the rare close passage of a star, which would cause a comet shower. But it turns out that even without a star encounter, long-period comets from the inner Oort Cloud can slip past the protective barrier posed by the presence of Jupiter and Saturn and travel a path that crosses Earth's orbit.

In the new research, Kaib and co-author Thomas Quinn, a UW astronomy professor and Kaib's doctoral adviser, used computer models to simulate the evolution of comet clouds in the solar system for 1.2 billion years. They found that even outside the periods of comet showers, the inner Oort Cloud was a major source of long-period comets that eventually cross Earth's path.

By assuming the inner Oort Cloud as the only source of long-period comets, they were able to estimate the highest possible number of comets in the inner Oort Cloud. The actual number is not known. But by using the maximum number possible, they determined that no more than two or three comets could have struck Earth during what is believed to be the most powerful comet shower of the last 500 million years.

"For the past 25 years, the inner Oort Cloud has been considered a mysterious, unobserved region of the solar system capable of providing bursts of bodies that occasionally wipe out life on Earth," Quinn said. "We have shown that comets already discovered can actually be used to estimate an upper limit on the number of bodies in this reservoir."

With three major impacts taking place nearly simultaneously, it had been proposed that the minor extinction event about 40 million years ago resulted from a comet shower. Kaib and Quinn's research implies that if that relatively minor extinction event was caused by a comet shower, then that was probably the most-intense comet shower since the fossil record began.

"That tells you that the most powerful comet showers caused minor extinctions and other showers should have been less severe, so comet showers are probably not likely causes of mass extinction events," Kaib said.

He noted that the work assumes the area surrounding the solar system has remained relatively unchanged for the last 500 million years, but it is unclear whether that is really the case. It is clear, though, that Earth has benefitted from having Jupiter and Saturn standing guard like giant catchers mitts, deflecting or absorbing comets that might otherwise strike Earth.

"We show that Jupiter and Saturn are not perfect and some of the comets from the inner Oort Cloud are able to leak through. But most don't," Kaib said.

Unexpected reservoir of monocytes discovered in the spleen

Mouse study indicates immune cells from spleen may be essential in healing heart attack damage

It takes a spleen to mend a broken heart – that's the conclusion of a surprising new report from researchers at the Massachusetts General Hospital (MGH) Center for Systems Biology, directed by Ralph Weissleder, MD, PhD. In the July 31 issue of Science the team reports how, in following up an intriguing observation, they discovered an unexpected reservoir of the immune cells called monocytes in the spleen and went on to show that these cells are essential to recovery of cardiac tissue in an animal heart attack model.

"Monocytes are known to serve as a central defense system against injury, and we found that monocytes released from the spleen go directly to the injured heart and participate in wound healing," says Matthias Nahrendorf, MD, PhD, a co-lead author of the study.

Monocytes are generated in the bone marrow, released into the blood and are known to accumulate at injured or infected tissues, where they differentiate into macrophages or dendritic cells. In investigating processes involved in the healing of ischemic heart tissue – the sort of injury produced in a heart attack – in mice, the research team was surprised to find more monocytes accumulating at the site of injury than would be found in the animals' entire circulatory system. When they searched many types of tissue for the presence of cells with monocyte-specific molecules, they only found significant numbers of such cells in the spleen.

Monocytes in the spleen were identical in appearance, composition and function to monocytes in the blood. To investigate the splenic monocyte reservoir's potential involvement in cardiac healing, the researchers used several new technologies. A newly developed microscopic technique allowed them to determine how and where monocytes are stored in the spleen – previously known to store red blood cells – and to study how monocytes are released in response to an experimentally-induced heart attack. A novel three-dimensional optical imaging technique (fluorescence molecular tomography, developed at the MGH Center for Molecular Imaging Research) allowed study of monocyte-mediated immune functions at the site of heart muscle injury.

In mice whose spleens were removed and replaced with a donor organ, an induced heart attack led to rapid increase of spleen-derived donor monocytes in the bloodstream and massive accumulation of donor cells at the site of injury. In animals from whom spleens were removed but not replaced, heart attack produced no significant monocyte increase in the bloodstream or in the heart. "With all these approaches together, we found that the monocytes that travel to the heart after a heart attack come directly from the spleen and that, without the splenic monocytes, the heart tissue does not heal well," says Filip Swirski, PhD, co-lead author of the Science report.

The investigators also found that the hormone angiotensin II, known to be released in response to a heart attack, is actively involved in the release of monocytes from the spleen. Identifying that pathway could lead to ways of manipulating the splenic monocyte reservoir to improve healing after a heart attack and potentially regulate other inflammatory situations. "We need to know whether this monocyte reservoir is important in other diseases – such as viral or bacterial infection, cancer or atherosclerosis – and understand how to precisely control storage and release of monocytes in a therapeutic setting, both of which we are currently investigating," says Mikael Pittet, PhD, senior author of the Science report.

Pittet and Nahrendorf are both assistant professors of Radiology at Harvard Medical School, and Swirski is an instructor in Radiology. Additional co-authors of the Science report are Martin Etzrodt, Moritz Wildgruber, Virna Cortez-Retamozo, Peter Panizzi, PhD, Jose-Luiz Figueiredo, MD, Rainer Kohler, PhD, Aleksey Chudnovskiy, Peter Waterman, Elena Aikawa, MD, PhD, Thorsten Mempel, MD, PhD, and Ralph Weissleder, MD, PhD, MGH Center for Systems Biology; and Peter Libby, MD, Brigham and Women's Hospital. The study was supported by grants from the National Institutes of Health and the MGH Center for Systems Biology.

Little lifesavers -- kids capable of CPR

Nine-year-olds can and should learn CPR. A study of 147 schoolchildren, published in BioMed Central's open access journal Critical Care, has shown that, although the smallest may lack the requisite strength, the knowledge of how to perform basic life support is well retained by young children.

Fritz Sterz, from the Medical University of Vienna, Austria, led a team of researchers who studied children who had received six hours of life support training. Upon examination four months after the training, 86% performed CPR correctly. Sterz said, "The usefulness of CPR training in schools has been questioned since young students may not have the physical and cognitive skills needed to perform such complex tasks correctly. We found that, in fact, students as young as 9 years are able to successfully and effectively learn basic life support skills. As in adults, physical strength may limit depth of chest compressions and ventilation volumes, but skill retention is good."

The skills taught to the children included automatic defibrillator deployment, providing CPR, usage of the recovery position and calling for the emergency services. For the critical skills of CPR and mouth-to-mouth

resuscitation, BMI was the factor that had the biggest influence on depth of compressions and amount of air inhaled. Age did not play a role, indicating that a well-built nine-year-old can be just as capable as an older child.

The researchers conclude, "Given the excellent performance by the students evaluated in this study, the data support the concept that CPR training can be taught and learnt by school children and that CPR education can be implemented effectively in primary schools at all levels. Even if physical strength may limit CPR effectiveness, cognitive skills are not dependent on age, and with periodic retraining, children's performance would likely improve over time."

Notes to Editors *1. School children sufficiently apply life supporting first aid: A prospective investigation. Roman Fleischhackl, Alexander Nuernberger, Fritz Sterz, Christina Schoenberg, Tania Urso, Tanja Habart, Martina Mittlboeck and Nisha Chandra-Strobos Critical Care (in press)*
During embargo, article available here: http://ccforum.com/imedia/8241666248271473_article.pdf?random=147924
After the embargo, article available at journal website: http://ccforum.com/

Got zinc? New zinc research suggests novel therapeutic targets

New report in the Journal of Leukocyte Biology suggests that zinc activates a key protein on T cells needed to fight infections

Everyone knows that vitamins "from A to zinc" are important for good health. Now, a new research study in the August 2009 print issue of the Journal of Leukocyte Biology (http://www.jleukbio.org) suggests that zinc may be pointing the way to new therapeutic targets for fighting infections. Specifically, scientists from Florida found that zinc not only supports healthy immune function, but increases activation of the cells (T cells) responsible for destroying viruses and bacteria.

"It has been shown that zinc supplementation significantly reduces the duration and severity of childhood diarrhea, lower respiratory infections, and incidence of malaria in zinc-deficient children," said report co-author, Robert Cousins, Ph.D., who also is the director of the Center for Nutritional Sciences within the Food Science and Human Nutrition Department at the University of Florida. "Age-related declines in immune function have also been related to zinc deficiency in the elderly."

Scientists administered either a zinc supplement or a placebo to healthy volunteers to assess the effects of zinc on T cell activation. After isolating the T cells from the blood, scientists then simulated infection in laboratory conditions. Results showed that T cells taken from the zinc-supplemented group had higher activation than those from the placebo group. Specifically, cell activation stimulated the zinc transporter in T cells called "ZIP8," which transports stored zinc into the cell cytoplasm where it then alters the expression of a T cell protein in a way needed to fight infections.

"As the debate over zinc supplementation in healthy individuals continues," said John Wherry, Ph.D., Deputy Editor of the Journal of Leukocyte Biology, "studies like this help shed light on how zinc may enhance the ability of our immune systems to fight off foreign invaders. Equally important, this work points toward new possible targets for entirely new drugs to help augment immune function and prevent or stop infections that might be resistant to traditional antibiotics."

Details: Tolunay B. Aydemir, Juan P. Liuzzi, Steve McClellan, and Robert J. Cousins Zinc transporter ZIP8 (SLC39A8) and zinc influence IFN- expression in activated human T cells. J Leukoc Biol 2009 86: 337.
http://www.jleukbio.org/cgi/content/abstract/86/2/337

Species barrier may protect macaques from chronic wasting disease

Data from an ongoing multi-year study suggest that people who consume deer and elk with chronic wasting disease (CWD) may be protected from infection by an inability of the CWD infectious agent to spread to people. The results to date show that 14 cynomolgus macaques exposed orally or intracerebrally to CWD remain healthy and symptom free after more than six years of observation, though the direct relevance to people is not definitive and remains under study. Cynomolgus macaques often are used as research models of human disease because they are very close genetically to humans and are susceptible to several forms of human brain-damaging disease. Thus, it was decided to see whether exposure to CWD could induce disease in the macaques. The study appears online in the journal Emerging Infectious Diseases.

CWD is a type of brain-damaging disease known as a transmissible spongiform encephalopathy (TSE) or prion disease. CWD primarily affects deer, elk, and moose. Other TSE diseases include mad cow disease, or bovine spongiform encephalopathy (BSE) in cattle, scrapie in sheep, and sporadic Creutzfeldt-Jakob disease (CJD) in humans. Humans are not susceptible to sheep scrapie, but BSE appears to have infected about 200 people, primarily in Europe in the 1990s. Those findings provided the rationale for the present CWD-macaque study, which began in 2003.

"We plan to continue this study for at least several more years because, although the risk to macaques so far appears to be low, we know that these diseases can take more than 10 years to develop," says Bruce Chesebro,

M.D., chief of the Laboratory of Persistent Viral Diseases at Rocky Mountain Laboratories (RML) in Hamilton, Mont. RML is part of the National Institute of Allergy and Infectious Diseases (NIAID) of the National Institutes of Health (NIH). The RML group is leading the study with collaborators from the Colorado Division of Wildlife; State University of New York Downstate Medical Center; New York State Institute for Basic Research in Developmental Disabilities; American Red Cross; and the University of Wyoming.

The findings by the RML group support published field studies done by others in regions of Colorado and Wyoming where CWD is endemic. Between 1979 and 2001, there were no significant increases in human TSE diseases despite the likelihood that hunters in those areas were exposed to CWD through contact with infected animal tissue and contaminated hunting tools such as knives and saws. Extensive laboratory data also supports a human species barrier against CWD.

Notably, the RML study also included identical testing in squirrel monkeys, which are genetically less similar to humans than macaques. Of 15 squirrel monkeys exposed orally to CWD, two displayed disease symptoms 69 months after infection. Of 13 squirrel monkeys exposed intracerebrally to CWD, 11 displayed symptoms between 33 and 53 months after infection. In symptomatic animals, the presence of the CWD agent was confirmed in brain, spleen and lymph nodes.

The results in squirrel monkeys were not surprising because a study elsewhere in two squirrel monkeys yielded similar results. The study by the RML group was different, however, in that it tested oral exposure to CWD and also studied eight CWD samples from different areas of the country. The results in squirrel monkeys confirmed that disease progression in that species appears consistent with disease progression in deer and elk, where severe weight loss is nearly always present.

"The fact that the squirrel monkeys, like the deer and elk, suffered severe weight loss suggests that chronic wasting disease might affect a common region of the brain in different species," notes Dr. Chesebro.

Reference: Race B et al. Susceptibilities of nonhuman primates to chronic wasting disease. Emerging Infectious Diseases. DOI: 10.3201/eid1509.090253.

Study Links Virus To Some Cases Of Common Skin CancerPosted 7/30/2009

COLUMBUS, Ohio – A virus discovered last year in a rare form of skin cancer has also been found in people with the second most common form of skin cancer among Americans, according to researchers at the Ohio State University Comprehensive Cancer Center - James Cancer Hospital and Solove Research Institute.

The researchers examined tissue samples from 58 people with squamous cell carcinoma (SCC), a highly curable form of skin cancer that is expected to affect more than 200,000 Americans this year.

They identified the virus in more than a third of the patients and in 15 percent of the tumors tested. In addition, all of the virus found in tumor cells had a mutation that could enable the viral DNA to integrate into the DNA of the host cell.

"This is indirect evidence that the virus might play a role in causing some cases of squamous cell carcinoma," says principal investigator Amanda E. Toland, assistant professor of molecular virology, immunology and medical genetics and a researcher with the Ohio State University Comprehensive Cancer Center - James Cancer Hospital and Solove Research Institute.

The findings are published in a recent issue of the Journal of Investigative Dermatology.

The virus was first discovered in patients with Merkel cell carcinoma, a rare, aggressive skin cancer that occurs mainly in the elderly and people with a suppressed immune system. The people in the new study all had a healthy immune system. "Originally it was thought that this virus caused only this rare skin cancer, but our findings indicate that it is a lot more prevalent than we initially thought."

To learn if people with SCC harbored the virus, Toland, working closely with first author and graduate research associate Amy Dworkin and Ohio State pathologists O. Hans Iwenofu and Sara B. Peters, examined DNA samples from SCC tumors, from normal-appearing skin adjacent to the tumor, when available; from white blood cells, and from cells washed from the mouth.

The investigators detected the virus in 26 of 177 SCC samples, 11 of 63 adjacent-skin samples, and one sample from a mouthwash. They found no viral DNA in any of the blood samples from 57 patients. In all, 21 of 58 SCC patients, or 36 percent, tested positive for the virus.

By sequencing the viral DNA from 31 normal and tumor samples, the researchers showed that the same mutation was present in all the viruses tested from tumors, and in 60 percent of the viruses tested from adjacent healthy-looking tissue. "That suggests that the virus may develop a mutation that causes it to integrate into host-cell DNA, and, therefore, may play a role in causing the cancer," Toland says.

Next, Toland wants to test normal skin in healthy individuals to learn how common this virus is in people generally and to learn whether the virus actually integrates with the host DNA.

"If it proves to be a cancer-causing virus, and if it proves to be common in the general population, it might be something we should begin screening people for," she says.

Funding from the American Cancer Society supported this research.

Ohio State researchers Stephanie Y. Tseng and Dawn C. Allain were also involved in this study.

The Ohio State University Comprehensive Cancer Center-James Cancer Hospital and Solove Research Institute (www.jamesline.com) is one of only 40 Comprehensive Cancer Centers in the United States designated by the National Cancer Institute. Ranked by U.S. News & World Report among the top 20 cancer hospitals in the nation, The James is the 180-bed adult patient-care component of the cancer program at The Ohio State University. The OSUCCC-James is one of only five centers in the country approved by the NCI to conduct both Phase I and Phase II clinical trials.

UF scientists program blood stem cells to become vision cells

University of Florida researchers were able to program bone marrow stem cells to repair damaged retinas in mice, suggesting a potential treatment for one of the most common causes of vision loss in older people.

The success in repairing a damaged layer of retinal cells in mice implies that blood stem cells taken from bone marrow can be programmed to restore a variety of cells and tissues, including ones involved in cardiovascular disorders such as atherosclerosis and coronary artery disease.

"To our knowledge, this is the first report using targeted gene manipulation to specifically program an adult stem cell to become a new cell type," said Maria B. Grant, M.D., a professor of pharmacology and therapeutics at UF's College of Medicine. "Although we used genes, we also suggest you can do the same thing with drugs - but ultimately you would not give the drugs to the patient, you would give the drugs to their cells. Take the cells out, activate certain chemical pathways, and put the cells back into the patient."

In a paper slated to appear in the September issue of the journal Molecular Therapy, scientists describe how they used a virus carrying a gene that gently pushed cultured adult stem cells from mice toward a fate as retinal cells. Only after the stem cells were reintroduced into the mice did they completely transform into the desired type of vision cells, apparently taking environmental cues from the damaged retinas.

After studying the cell-transformation process, scientists were able to bypass the gene manipulation step entirely and instead use chemical compounds that mirrored environmental conditions in the body, thus pointing the stem cells toward their ultimate identities as vision cells.

"First we were able to show you can overexpress a protein unique to a retinal cell type and trick the stem cell into thinking it is that kind of cell," said Grant, who collaborated with Edward Scott, Ph.D., the director of the Program in Stem Cell Biology and Regenerative Medicine at UF's McKnight Brain Institute. "As we proceeded, we found we could activate the stem cells by mimicking the body's natural signaling channels with chemicals. This implies a whole new field of stem cell research that uses drug manipulation rather than genetic manipulation to send these immature cells along new pathways."

Scientists chose to build retinal pigment epithelial cells, which form the outer barrier of the retina. In addition to being very specialized and easy to identify, RPE cells are faulty in many retinal diseases, including age-related macular degeneration, which affects nearly 2 million people in the United States, and some forms of blindness related to diabetes.

"This work applies to 85 percent of patients who have age-related macular degeneration," Grant said. "There are no therapies for this devastating disease."

The work was supported by the National Eye Institute. Researchers removed blood stem cells from the bone marrow of mice, modified the cells in cultures, and injected them back into the animals' circulatory systems. From there, the stem cells were able to home in on the eye injury and become retinal cells.

At 28 days after receiving the modified stem cells, mice that had previously demonstrated no retinal function were no different than normal mice in electrical measures of their response to light.

Grant and UF have patented some technology involved in the research.

From graphene to graphane, now the possibilities are endless

Ever since graphene was discovered in 2004, this one-atom thick, super strong, carbon-based electrical conductor has been billed as a "wonder material" that some physicists think could one day replace silicon in computer chips.

But graphene, which consists of carbon atoms arranged in a honeycomb lattice, has a major drawback when it comes to applications in electronics – it conducts electricity almost too well, making it hard to create graphene-based transistors that are suitable for integrated circuits.

In August's Physics World, Kostya Novoselov - a condensed-matter physicist from the Manchester University group that discovered graphene -- explains how their discovery of graphane, an insulating equivalent of graphene, may prove more versatile still.

Graphane has the same honeycomb structure as graphene, except that it is "spray-painted" with hydrogen atoms that attach themselves to the carbon. The resulting bonds between the hydrogen and carbon atoms effectively tie down the electrons that make graphene so conducting. Yet graphane retains the thinness, super-strength, flexibility and density of its older chemical cousin.

One advantage of graphane is that it could actually become easier to make the tiny strips of graphene needed for electronic circuits. Such structures are currently made rather crudely by taking a sheet of the material and effectively burning away everything except the bit you need. But now such strips could be made by simply coating the whole of a graphene sheet – except for the strip itself - with hydrogen. The narrow bit left free of hydrogen is your conducting graphene strip, surrounded by a much bigger graphane area that electrons cannot go down.

As if this is not enough, the physicists in Manchester have found that by gradually binding hydrogen to graphene they are able to drive the process of transforming a conducting material into an insulating one and watch what happens in between.

Perhaps most importantly of all, the discovery of graphane opens the flood gates to further chemical modifications of graphene. With metallic graphene at one end and insulating graphane at the other, can we fill in the divide between them with, say, graphene-based semiconductors or by, say, substituting hydrogen for fluorine?

As Professor Novoselov writes, "Being able to control the resistivity, optical transmittance and a material's work function would all be important for photonic devices like solar cells and liquid-crystal displays, for example, and altering mechanical properties and surface potential is at the heart of designing composite materials. Chemical modification of graphene – with graphane as its first example – uncovers a whole new dimension of research. The capabilities are practically endless."

Dementia induced and blocked in Parkinson's fly model

St. Louis, July 31, 2009 — Parkinson's disease is well-known for impairing movement and causing tremors, but many patients also develop other serious problems, including sleep disturbances and significant losses in cognitive function known as dementia.

Now researchers at Washington University School of Medicine in St. Louis have modeled Parkinson's-associated dementia for the first time. Scientists showed that a single night of sleep loss in genetically altered fruit flies caused long-lasting disruptions in the flies' cognitive abilities comparable to aspects of Parkinson's-associated dementia. They then blocked this effect by feeding the flies large doses of the spice curcumin.

"Clinical trials of curcumin to reduce risk of Parkinson's disease are a future possibility, but for now we are using the flies to learn how curcumin works," says author James Galvin, M.D., a Washington University associate professor of neurology who treats patients at Barnes-Jewish Hospital. "This should help us find other compounds that can mimic curcumin's protective effects but are more specific."

Galvin and senior author Paul Shaw, Ph.D., assistant professor of neurobiology, publish their results in the journal Sleep on Aug. 1.

Galvin is an expert in cognitive impairments in human Parkinson's disease; Shaw studies sleep and the brain in fruit flies. The researchers decided collaborate based in part on evidence that increased sleep loss in Parkinson's patients can precede or coincide with increased severity in other Parkinsonian symptoms.

More than 74 percent of Parkinson's patients have trouble sleeping, and up to 80 percent of patients 65 and older who have Parkinson's disease for seven years will develop dementia, according to Galvin.

Shaw's lab has linked sleep loss to changes in the dopaminergic system of the brain, the part of the brain that produces the neurotransmitter dopamine and is at the center of the damage caused by Parkinson's.

"In healthy flies, sleep deprivation decreases dopamine receptor production and causes temporary learning impairments that are fully restored after a two-hour nap," Shaw says.

Shaw and Galvin studied fruit flies genetically modified to make a human protein called alpha-synuclein in their brains. Scientists don't yet know what alpha-synuclein does, nor have they found a fly counterpart for it. But they have shown that it aggregates in the brains of Parkinson's disease patients and believe the processes that cause the aggregations are harming dopamine-producing cells.

Prior studies of fruit flies with human alpha-synuclein in their brains showed that the flies, like human Parkinson's patients, also lose dopamine-producing neurons, have movement-related problems and develop alpha-synuclein aggregations. But scientists had yet to evaluate the flies for signs of dementia.

Lead author Laurent Seugnet, Ph.D., research associate at L'Ecole Supérieure de Physique Chimie Industrielles in France, first tested the flies' learning ability using a procedure he helped develop in Shaw's lab. For the test, Seugnet placed flies in a vial with two branches: one lighted branch containing quinine, a bitter-

tasting substance flies prefer to avoid; and a darkened but quinine-free branch. After a few trials, normal flies learn to suppress their natural attraction to the light and fly into the darkened vial instead to avoid the quinine.

Flies with alpha-synuclein in their brains could still learn when they were middle-aged, or about 16 to 20 days old. But when Seugnet deprived them of sleep for 12 hours, he found that their ability to remember was more severely impaired than that of young healthy flies that had also been sleep-deprived.

"This was still true even 10 days later, so it seemed to be a lasting effect," says Seugnet.

Galvin had earlier found that curcumin, a derivative of the spice turmeric, blocks alpha-synuclein aggregation in cell models of Parkinson's disease. Based on this, Seugnet fed curcumin to a new batch of flies, repeated the tests and found middle-aged flies with alpha-synuclein retained their ability to learn as well as normal young flies.

"Thanks to this model our labs have created, Dr. Galvin and I can not only quickly test potential new treatments for these symptoms of Parkinson's, we can also move up our treatments in terms of the timeline along which the disorder develops," says Shaw. "That may give us a real chance to change the course of the disease."

Seugnet L, Galvin JE, Suzuki Y, Gottschalk L, Shaw PJ. Persistent short-term memory defects following sleep deprivation in a Drosophila model of Parkinson disease. Sleep, Aug. 1, 2009

Gorilla HIV makes leap to humans

* 18:00 02 August 2009 **by Ewen Callaway**

A new strain of HIV has jumped from gorillas to humans. So far, only one person, a 62-year-old French woman from Cameroon, has been found to be infected with the virus, which closely resembles strains of simian immunodeficiency virus (SIV) recently discovered in western gorillas in the wild.

"It would be surprising if there aren't some more" human cases, says David Robertson, a bioinformaticist at the University of Manchester, UK who analysed the virus's DNA along with colleagues in France. "We don't think this is a direct gorilla-to-human transmission."

Drug hope

Until 2004, the infected woman lived in a suburb of Cameroon's capital city Yaoundé, where she didn't come into contact with apes or eat their meat – SIV's primary route to humans. This means that she probably acquired the infection from another human, likely through sexual contact.

The woman hasn't yet shown any sign of a compromised immune system – the hallmark of AIDS – but tests on laboratory-cultured human cells suggest that the virus can replicate in the same white blood cells as other strains of HIV. However, the new virus should be susceptible to anti-retroviral drugs that slow the growth of other strains of HIV, Robertson says.

Based on its genetic sequence, the virus appears most closely related to a number of SIV strains collected recently in gorilla faecal samples from forests in Cameroon. These viruses also resemble "group O" strains of HIV, which infect far fewer people than other strains of HIV, most of them in Cameroon.

Original source

The discovery of a gorilla virus in humans could suggest that some of these group O viruses came from gorillas, Robertson says, but it's also possible that the virus originated chimpanzees and was transmitted independently to gorillas and to humans.

"Until we do more sampling we're also guessing a little bit," Robertson concedes.

Moreover, the virus's discovery adds to growing evidence that new HIV strains may regularly emerge from other primates, says Martine Peeters, a virologist at the University of Montpellier, who led the team that identified the first gorilla SIV strains. "It is just an additional demonstration that these viruses have jumped several times from apes to humans."

Virologists could be missing many of these transmissions because existing HIV tests are biased to identify already circulating strains of the virus. "We're very good at detecting HIV, so unless you do these other more in depth tests, you wouldn't necessarily detect these very divergent viruses," Robertson says.

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