Limit sucrose as painkiller for newborns

Randomized controlled trial

Using sucrose to reduce pain in newborns undergoing painful procedures should be limited to babies having blood taken (venipuncture) for the newborn screening test but not for intramuscular injections, write Dr. Anna Taddio and co-authors.

In this double-blind, randomized controlled trial of 240 newborns at Toronto's Mount Sinai Hospital, researchers found that "sucrose reduced overall pain in newborns when administered before painful medical procedures during the first 2 days after birth." However, "unexpectedly, we did not observe analgesic effects during either intramuscular injection of vitamin K in either group [newborns of diabetic and nondiabetic mothers] or during repeated heel-lancing for blood glucose monitoring in newborns of diabetic mothers."

As all newborns experience pain from medical procedures in the first days of life, the results of this study will be helpful in reducing pain responses. The authors recommend updating pain management guidelines to reflect these findings.

When using gestures, rules of grammar remain the same

The mind apparently has a consistent way of ordering an event that defies the order in which subjects, verbs, and objects typically appear in languages, according to research at the University of Chicago.

"Not surprisingly, speakers of different languages describe events using the word orders prescribed by their language. The surprise is that when the same speakers are asked to 'speak' with their hands and not their mouths, they ignore these orders – they all use exactly the same order when they gesture," said Susan Goldin-Meadow, the Bearsdley Rum Distinguished Service Professor in Psychology and lead author of the paper, "The Natural Order of Events: How Speakers of Different Languages Represent Events Nonverbally" published in the current issue of the Proceedings of the National Academy of Sciences.

For the study, the team tested 40 speakers of four different languages: 10 English, 10 Mandarin Chinese, 10 Spanish and 10 Turkish speakers. They showed them simple video sequences of activities and asked them to describe the action first in speech and a second time using only gestures. They also gave another 40 speakers of the same languages transparencies to assemble after watching the video sequences. Some of the videos portrayed real people and others animated toys that represented a variety of sentence types: a girl waves, a duck moves to a wheelbarrow, a woman twists a knob and a girl gives a flower to man.

When asked to describe the scenes in speech, the speakers used the word orders typical of their respective languages. English, Spanish, and Chinese speakers first produced the subject, followed by the verb, and then the object (woman twists knob). Turkish speakers first produced the subject, followed by the object, and then the verb (woman knob twists).

But when asked to describe the same scenes using only their hands, all of the adults, no matter what language they spoke, produced the same order — subject, object, verb (woman knob twists). When asked to assemble the transparencies after watching the video sequences (another nonverbal task, but one that is not communicative), people also tended to follow the subject, object, verb ordering found in the gestures produced without speech.

The grammars of modern languages developed over time and are the result of very distant cultural considerations that are difficult for linguists to study.

Newly emerging sign languages, however, offer intriguing corroborating evidence that the subject-object-verb (SOV) order is a fundamental one.

SOV is the order currently emerging in a language created spontaneously without any external influence. Al-Sayyid Bedouin Sign Language arose within the last 70 years in an isolated community with a high incidence of profound prelingual deafness. In the space of one generation, the language assumed grammatical structure, including the SOV order.

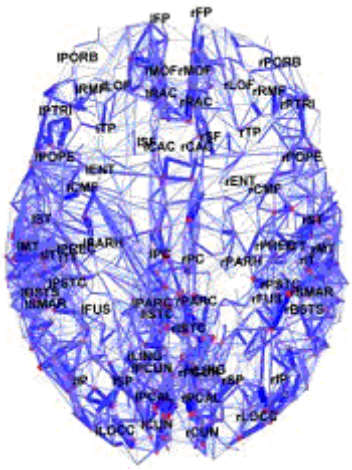
Moreover, when deaf children invent their own gesture systems, they use OV order. Chinese and American deaf children, whose hearing losses prevent them from acquiring spoken language and whose hearing parents have not exposed them to sign language, use the OV order in the gesture sentences they create.

The research challenges the idea that the language we speak inevitably shapes the way we think when we are not speaking. This study is the first to test the notion with respect to word order.

"Our data suggest that the ordering we use when representing events in a nonverbal format is not highly susceptible to language's influence," Goldin-Meadow and her co-authors write. "Rather, there appears to be a natural order that humans use when asked to represent events nonverbally. Indeed, the influence may well go in the other direction—the ordering seen in our nonverbal tasks may shape language in its emerging stages."

Joining Goldin-Meadow in writing the paper were Wing Chee So, of the National University of Singapore; Ali Ozyurek, of Radboud Universtiy Nijmegen, and Carolyn Mylander, a researcher at the University of Chicago.

New map IDs the core of the human brain



BLOOMINGTON, Ind. -- An international team of researchers has created the first complete high-resolution map of how millions of neural fibers in the human cerebral cortex -- the outer layer of the brain responsible for higher level thinking -- connect and communicate. Their groundbreaking work identified a single network core, or hub, that may be key to the workings of both hemispheres of the brain.

The work by the researchers from Indiana University, University of Lausanne, Switzerland, Ecole Polytechnique Fédérale de Lausanne, Switzerland, and Harvard Medical School marks a major step in understanding the most complicated and mysterious organ in the human body. It not only provides a comprehensive map of brain connections (the brain "connectome"), but also describes a novel application of a non-invasive technique that can be used by other scientists to continue mapping the trillions of neural connections in the brain at even greater resolution, which is becoming a new field of science termed "connectomics."

The first complete high-resolution map of the human cerebral cortex identifies a single network core that could be key to the workings of both hemispheres of the brain. **Indiana University**

"This is one of the first steps necessary for building large-scale computational models of the human brain to help us understand processes that are difficult to observe, such as disease states and recovery processes to injuries," said Olaf Sporns, co-author of the study and neuroscientist at Indiana University.

The findings appear in the journal PLoS Biology today (June 30). Co-authors include Patric Hagmann and Reto Meuli, University Hospital Center and University of Lausanne; Leila Cammoun and Xavier Gigandet, Ecole Polytechnique Fédérale de Lausanne; Van J. Wedeen, Massachusetts General Hospital and Harvard Medical Center; and Christopher J. Honey, IU.

Until now, scientists have mostly used functional magnetic resonance imaging (fMRI) technology to measure brain activity -- locating which parts of the brain become active during perception or cognition -- but there has been little understanding of the role of the underlying anatomy in generating this activity. What is known of neural fiber connections and pathways has largely been learned from animal studies, and so far, no complete map of brain connections in the human brain exists.

In this new study, a team of neuroimaging researchers led by Hagmann used state-of-the-art diffusion MRI technology, which is a non-invasive scanning technique that estimates fiber connection trajectories based on gradient maps of the diffusion of water molecules through brain tissue. A highly sensitive variant of the method, called diffusion spectrum imaging (DSI), can depict the orientation of multiple fibers that cross a single location. The study applies this technique to the entire human cortex, resulting in maps of millions of neural fibers running throughout this highly furrowed part of the brain.

Sporns then carried out a computational analysis trying to identify regions of the brain that played a more central role in the connectivity, serving as hubs in the cortical network. Surprisingly, these analyses revealed a single highly and densely connected structural core in the brain of all participants.

"We found that the core, the most central part of the brain, is in the medial posterior portion of the cortex, and it straddles both hemispheres," Sporns said. "This wasn't known before. Researchers have been interested in this part of the brain for other reasons. For example, when you're at rest, this area uses up a lot of metabolic energy, but until now it hasn't been clear why."

The researchers then asked whether the structural connections of the brain in fact shape its dynamic activity, Sporns said. The study examined the brains of five human participants who were imaged using both fMRI and DSI techniques to compare how closely the brain activity observed in the fMRI mapped to the underlying fiber networks.

"It turns out they're quite closely related," Sporns said. "We can measure a significant correlation between brain anatomy and brain dynamics. This means that if we know how the brain is connected we can predict what the brain will do."

Sporns said he and Hagmann plan to look at more brains soon, to map brain connectivity as brains develop and age, and as they change in the course of disease and dysfunction.

The study can be viewed at http://www.plos.org/press/plbi-06-07-sporns.pdf. After June 30, the article can be viewed at http://biology.plosjournals.org/perlserv/?request=get-document&doi=10.1371/journal.pbio.0060159.

The study was supported in part by the J.S. McDonnell Foundation, the University of Lausanne, Center for Biomedical Imaging (CIBM) of the Geneva-Lausanne Universities, Ecole Polytechnique Fédérale de Lausanne and the National Institutes of Health.

Fortified cassava could provide a day's nutrition in a single meal

COLUMBUS, Ohio – Scientists have determined how to fortify the cassava plant, a staple root crop in many developing countries, with enough vitamins, minerals and protein to provide the poor and malnourished with a day's worth of nutrition in a single meal.

The researchers have further engineered the cassava plant so it can resist the crop's most damaging viral threats and are refining methods to reduce cyanogens, substances that yield poisonous cyanide if they are not properly removed from the food before consumption. The reduction of cyanogens also can shorten the time it takes to process the plant into food, which typically requires three to six days to complete.

Studies also are under way to extend the plant's shelf life so it can be stored or shipped.

The international team of scientists hopes to translate the greenhouse research into a product that can be field tested in at least two African nations by 2010. Funded by more than $12.1 million in grants from the Bill & Melinda Gates Foundation, the group of researchers is led by Richard Sayre, a professor of plant cellular and molecular biology at Ohio State University.

Sayre presented an update on the BioCassava Plus project June 30 at the American Society of Plant Biologists meeting in Mérida, Mexico.

"This is the most ambitious plant genetic engineering project ever attempted," Sayre said. "Some biofortification strategies have the objective of providing only a third of the daily adult nutrition requirements since consumers typically get the rest of their nutritional requirements from other foods in their diet. But global food prices have recently gone sky high, meaning that many of the poorest people are now eating just one meal a day, primarily their staple food.

"So what we're working on has become even more important in the last year than it was when we started, not just in regions where people are malnourished, but across developing countries where food has gotten so expensive that people can't afford the diverse diet that they're used to."

Cassava (Manihot esculenta) is the primary source of calories for an estimated 800 million people worldwide, including 250 million people in sub-Saharan Africa, the current focus of the Gates-funded project. But the plentiful crop has several drawbacks. It is composed almost entirely of carbohydrates so it does not provide complete nutrition.

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The roots can be banked in the ground for up to three years, providing food security, but the plant must undergo time-consuming processing immediately after harvest to remove compounds that generate cyanide. Unprocessed roots also deteriorate within 48 hours after harvest, limiting the food's shelf life. And a plant disease caused by the geminivirus reduces yields by 30 percent to 50 percent in many areas in sub-Saharan Africa, a major blow to farm productivity.

Sayre and colleagues from multiple institutions set out to tackle virtually all of cassava's problems to make the plant more nutritious and to increase the crop's revenue-producing potential for farmers.

Sayre reported that the research team has been able to address each of the plant's deficiencies in individual transgenic plants. The next step will be to combine some or all of the bioengineered traits into a single, farmer-preferred cultivar, with the goal of eventually developing cassava varieties that carry all of the improvements developed by the researchers.

"We've begun field trials in Puerto Rico to make sure the plants perform as well outside as they do in greenhouses, and we hope to start field trials in the target countries of Nigeria and Kenya by 2009," Sayre said.

The labs in the project have used a variety of techniques to improve on the model cassava plant used for the research. They used genes that facilitate mineral transport to produce a cassava root that accumulates more iron and zinc from the soil. To fortify the plants with a form of vitamin E and beta-carotene (also called pro-vitamin A because it converts to vitamin A in the body), the scientists introduced genes into the plant that increase terpenoid and carotenoid production, the precursors for pro-vitamin A and vitamin E. They achieved a 30-fold increase in pro-vitamin A, which is critical for human vision, bone and skin health, metabolism and immune function.

Adding protein to the cassava plant has posed a challenge, Sayre said. The scientists discovered that most of the nitrogen required to make the amino acids used for protein synthesis in roots is derived from the cyanogens that also cause cyanide toxicity. So their strategy for increasing protein levels in roots focuses on accelerating the conversion of cyanide-containing compounds into protein rather than completely eliminating cyanogen production, which would hinder the efforts to increase protein production, Sayre explained. To further address the cyanide problem, the scientists have also developed a way to accelerate the processing methods required to

remove cyanide – a days-long combination of peeling, soaking and drying the roots before they are eaten.

To strengthen the cassava plant's resistance to viruses, the scientists introduced a protein and small interfering RNA molecules that interfere with the viruses' ability to reproduce.

Prolonging cassava's shelf life has involved the development of a hybrid species that crosses two related plants native to Texas and Brazil. The strategy, still in development, will combine the properties of these plants and additional genes that function as antioxidants, slowing the rotting process that has been traced to the production of free radicals that damage and kill cells in newly harvested cassava roots.

The first cassava product the team plans to develop for investigations in the field will likely include the virus resistance, elevated protein, elevated beta-carotene (pro-vitamin A) and elevated minerals (iron and zinc), Sayre said.

"These traits have been working the best in the greenhouse, and the virus resistance is critical to success in the field," he said. "The thinking behind starting with these four traits is driven by science and by the impact they can have."

The BioCassava Plus project was launched with a $7.5 million grant from the Gates Foundation and recently received an additional $4.6 million in supplemental funding from the foundation to accelerate the application of this research in Africa by African scientists. The supplemental funding will support the training of African scientists so they can produce the transgenic plants in African institutions for use on African farms.

"It will not only be an improved staple crop eaten as a main source of nutrition, but we're also looking at the transformation of cassava from a staple crop to an income-generating crop," Sayre said. "That lifts people out of poverty, allows families to send kids to school and build infrastructure in their villages, so this is an important way to cross cultural barriers. There are many different cultures and languages in Africa, but higher crop yield, productivity, longer shelf life and making money are things that everyone understands."

The BioCassava Plus research team includes Claude Fauquet, Nigel Taylor, Dan Shachtman, Ed Cahoon and Paul Anderson of the Donald Danforth Plant Science Center in St. Louis; Willi Gruissem and Peng Zhang of the Swiss Federal Institute of Technology in Zurich; John Beeching of the University of Bath in England; John Fellman of Washington State University; Martin Fregene and Hernan Ceballos of the International Center for Tropical Agriculture in Colombia; Ivan Ingelbrecht, Alfred Dixon and Bussie Maziya-Dixon of IITA-Nigeria (an African research organization); Caroline Herron of IITA-Kenya; Simon Gichuki of the Kenya Agricultural Research Institute; Ada Mbanaso of the National Root Crops Research Institute in Nigeria; Dimuth Siritunga of the University of Puerto Rico; Mark Manary of Washington University; and independent consultant Jeff Stein. Mary Ann Abiado and Kristen Mosier of Ohio State provide administrative oversight.

Low levels of good cholesterol linked to memory loss, dementia risk

American Heart Association rapid access journal report

Low levels of high-density lipoproteins (HDL) — the "good" cholesterol — in middle age may increase the risk of memory loss and lead to dementia later in life, researchers reported in Arteriosclerosis, Thrombosis and Vascular Biology: Journal of the American Heart Association.

Observing 3,673 participants (26.8 percent women) from the Whitehall II study, researchers found that falling levels of HDL cholesterol were predictors of declining memory by age 60. Whitehall II, which began in 1985, is long-term health examination of more than 10,000 British civil servants working in London.

"Memory problems are key in the diagnosis of dementia," said Archana Singh-Manoux, Ph.D., lead author of the study and Senior Research Fellow with the French National Institute for Health and Medical Research (INSERM, France) and the University College London in England. "We found that a low level of HDL may be a risk factor for memory loss in late midlife. This suggests that low HDL cholesterol might also be a risk factor for dementia."

Researchers defined low HDL as less than 40 mg/dL and high HDL as 60 mg/dL or higher. The team compared blood-fat and memory data collected in phases 5 (1995?) and 7 (2002?) of Whitehall II, when the average ages of the study members were 55 and 61 years, respectively.

Researchers measured lipid concentrations in blood samples collected after an eight-hour fast, or at least four hours after a light, fat-free breakfast. They assessed short-term verbal memory using 20 one- or two-syllable words read aloud at two-second intervals. Study participants then had two minutes to write down as many of the words as they could remember.

Their main findings are:

* At age 55, participants with low HDL cholesterol showed a 27 percent increased risk of memory loss when compared to those with high HDL.

* At age 60, participants with low HDL had a 53 percent increased risk of memory loss compared to the high HDL group.

* During the five years between phases 5 and 7, study members with decreasing HDL had a 61 percent increased risk of decline in their ability to remember words versus those with high HDL.

* Men and women did not differ significantly in the link between lipids and memory loss, so researchers combined data from both sexes for analysis.

* Total cholesterol and triglycerides did not show a link with memory decline.

* Using statin drugs to raise HDL and/or lower low-density lipoprotein (LDL or "bad" cholesterol) showed no association with memory loss.

HDL cholesterol, which at high levels decreases the risk of heart attacks, serves several vital biological functions. It helps clear excess cholesterol from the blood; assists nerve-cell synapses to mature; and helps control the formation of beta-amyloid, the major component of the protein plaques found in the brains of Alzheimer's patients. Dementia most often occurs in people 65 years or older, the fastest growing age group in the industrialized world.

The precise mechanism linking HDL cholesterol to dementia remains unclear. "But it is possible that HDL cholesterol prevents formation of beta-amyloid," Singh-Manoux said. "HDL could also affect memory through its influence on atherosclerotic disease and stroke, or vascular injury. Finally, HDL cholesterol may influence memory through its anti-inflammatory and antioxidant effects.

"Many previous investigations into the association between lipids and memory in the elderly have focused on total or LDL cholesterol because of their status as proven risk factors for cardiovascular disease," Singh-Manoux said. "Our results show HDL cholesterol to be important for memory. Thus, physicians and patients should be encouraged to monitor levels of HDL cholesterol."

In an accompanying editorial, Anatol Kontush, Ph.D. and M. John Chapman, Ph.D., D.Sc., at INSERM and University Pierre and Marie Curie in Paris, France, note, "It is tempting to speculate that increasing levels of HDL-C, or "good cholesterol" might protect our memories. However, unfortunate results in large interventional trials with dietary antioxidants suggest that we should remain cautious when proposing therapeutic intervention on the basis of observational studies which do not allow causation to be inferred. Nonetheless, these studies demand that we focus more effort on research at the interface between HDL and brain function."

To raise HDL and lower LDL cholesterol, the American Heart Association recommends exercising regularly; eliminating trans fats from the diet; reducing the intake of all fats, especially saturated fats; and consuming monounsaturated fats, such as olive, canola and peanut oils. Statins can also improve HDL and LDL cholesterol levels, when they pose a heart risk.

Co-authors are: David Gimeno, Ph.D.; Mika Kivimaki, Ph.D.; Eric Brunner, Ph.D.; and Michael G. Marmot, M.D., Ph.D.

Happiness is rising around the world: U-M study

ANN ARBOR, Mich.---People in most countries around the world are happier these days, according to newly released data from the World Values Survey based at the University of Michigan Institute for Social Research.

Data from representative national surveys conducted from 1981 to 2007 show the happiness index rose in an overwhelming majority of nations studied.

"It's a surprising finding," said U-M political scientist Ronald Inglehart, who directs the World Values Surveys and is the lead author of an article on the topic to be published in the July 2008 issue of the journal Perspectives on Psychological Science. "It's widely believed that it's almost impossible to raise an entire country's happiness level."

The 2007 wave of the surveys also provides a ranking of 97 nations containing 90 percent of the world's population. The results indicate that Denmark is the happiest nation in the world and Zimbabwe the unhappiest. The United States ranks 16th on the list, immediately after New Zealand.

During the past 26 years, the World Values Surveys have asked more than 350,000 people how happy they are, using the same two questions.

"Taking all things together, would you say you are very happy, rather happy, not very happy, not at all happy?" And, "All things considered, how satisfied are you with your life as a whole these days?"

Combining responses to these two questions, Inglehart and colleagues constructed an index of subjective well-being that reflects both happiness and general life satisfaction.

In the 52 countries for which a substantial time series is available (covering 17 years on average), this index rose in 40 countries and fell in only 12. The average percentage of people who said they were "very happy" increased by almost seven points.

"Most earlier research has suggested that happiness levels are stable," Inglehart said. "Important events like winning the lottery or learning you have cancer can lead to short-term changes, but in the long run most previous research suggests that people and nations are stuck on a 'hedonic treadmill.' The belief has been that no matter what happens or what we do, basic happiness levels are stable and don't really change."

The new findings from the World Values Surveys not only show that during the past 25 years, happiness has in fact risen substantially in most countries. Fully as important as the fact that happiness rose is the reason why.

In recent decades, low-income countries such as India and China have experienced unprecedented rates of economic growth, dozens of medium-income countries have democratized and there has been a sharp rise of gender equality and tolerance of ethnic minorities and gays and lesbians in developed societies.

Economic growth, democratization and rising social tolerance have all contributed to rising happiness, with democratization and rising tolerance having even more impact than economic growth. All of these changes have contributed to providing people with a wider range of choice in how to live their lives---which is a key factor in happiness.

The people of rich countries tend to be happier than those of poor countries, but even controlling for economic factors, certain types of societies are much happier than others.

"The results clearly show that the happiest societies are those that allow people the freedom to choose how to live their lives," Inglehart said.

As an example, Inglehart points to the tolerant social norms and democratic political systems in Denmark, Iceland, Switzerland, the Netherlands and Canada all of which rank among the 10 happiest countries in the world.

"The events of the past 25 years have brought a growing sense of freedom that seems to be even more important than economic development in contributing to rising happiness," Inglehart said. "Moreover, the most effective way to maximize happiness seems to change with rising levels of economic development. In subsistence-level societies, happiness is closely linked with in-group solidarity, religiosity and national pride. At higher levels of economic security, free choice has the largest impact on happiness."

He also notes that the largest recent increases on the subjective well-being index, measuring both happiness and life-satisfaction, occurred in the Ukraine, followed by Moldova, Slovenia, Nigeria, Turkey and Russia.

"While most ex-communist countries show low levels of happiness, many of them show large recent increases in subjective well-being," Inglehart said. "The collapse of communism was generally followed by a sharp decline in well-being, which tended to rise again with economic recovery."

Comparing World Values Survey data from 1981 to 2007 with earlier data from 1946 from the World Database of Happiness, Inglehart and colleagues found that 19 of 24 countries show rising happiness and several countries---India, Ireland, Mexico, Puerto Rico and South Korea---show steeply rising trends. Only four countries show downward trends---Austria, Belgium, the United Kingdom and West Germany. For more information on happiness trends in more than 20 nations, visit: http://www.worldvaluessurvey.org/happinesstrends

The World Values Surveys in the United States and in several other countries are funded by the National Science Foundation. Additional funding for the surveys comes from a variety of agencies and foundations around the world, including the Swedish and Netherlands Foreign Ministries.

For more information on the World Values Survey: www.worldvaluessurvey.org

Researchers link early stem cell mutation to autism

La Jolla, Calif., June 30, 2008--In a breakthrough scientific study published today in the Proceedings of the National Academy of Sciences, scientists at the Burnham Institute for Medical Research have shown that neural stem cell development may be linked to Autism. The study demonstrated that mice lacking the myocyte enhancer factor 2C (MEF2C) protein in neural stem cells had smaller brains, fewer nerve cells and showed behaviors similar to those seen in humans with a form of autism known as Rett Syndrome.

This work represents the first direct link between a developmental disorder of neural stem cells and the subsequent onset of autism.

The research team was led by Stuart A. Lipton, M.D., Ph.D., a clinical neurologist and Professor and Director of the Del E. Webb Neuroscience, Aging and Stem Cell Research Center at Burnham.

"These results give us a good hint of how to look at Rett Syndrome and potentially other forms of autism in humans," said Dr. Lipton. "Having identified a mutation that causes this defect, we can track what happens. Perhaps we can correct it in a mouse, and if so, eventually correct it in humans."

Discovered in Dr. Lipton's laboratory, MEF2C turns on specific genes which drive stem cells to become nerve cells. When MEF2C was deleted from neural stem cells in mice, there was a faulty distribution of neurons accompanied by severe developmental problems. Adult mice lacking MEF2C in their brains displayed abnormal anxiety-like behaviors, decreased cognitive function and marked paw clasping, a behavior which may be analogous to hand wringing, a notable feature in humans with Rett syndrome.

"There's a yin and yang to this MEF2C protein," said Dr. Lipton. "My laboratory recently showed that MEF2C induces embryonic stem cells to become neurons. In this new research, we show that knocking out MEFC2 in the brain results in mice with smaller brains, fewer neurons and reduced neuronal activity. The commonality is the protein's association in making new neurons."

Collaborators were Drs. Hao Li, Shu-ichi Okamoto, Nobuki Nakanishi and Scott McKercher, of Burnham, as well as Dr. Amanda Roberts from The Scripps Research Institute and Dr. John Schwarz from the Albany Medical Center.

Rett syndrome, a form of autism, afflicts more girls than boys and results in poor brain development, repetitive hand motions, altered anxiety behaviors and the inability to speak. Patients with Rett Syndrome also suffer from seizures and other debilitating neurological symptoms.

Transplant drug may ease disorder linked to autism

A DRUG that prevents immune rejection in human transplant patients has improved the memory of mice with a hereditary learning disorder. The finding suggests the disorder is a result of reversible abnormalities in brain chemistry rather than irreversible differences in architecture, as was previously assumed.

Tuberous sclerosis complex (TSC) is a genetic disease that causes memory and learning problems. The drug rapamycin is known to interact with an enzyme that makes proteins needed for memory - as well as suppressing immune cells - so Alcino Silva and his colleagues at the University of California, Los Angeles, wondered if it might have an effect on TSC. Within three days of injecting it into mice with a version of TSC, the animals were as good as normal mice at finding submerged escape platforms and hidden food in mazes (Nature Medicine, DOI: 10.1038/nm1788).

Trials are now under way in the UK to test rapamycin in people with TSC. Half of those with TSC develop autism, but whether rapamycin "would be relevant or even desirable" for people with autism is not yet clear, says Simon Baron-Cohen of the Autism Research Centre at the University of Cambridge.

Watermelon May Have Viagra-Effect

Secrets of Phyto-nutrients Are Being Unraveled

COLLEGE STATION -- A cold slice of watermelon has long been a Fourth of July holiday staple. But according to recent studies, the juicy fruit may be better suited for Valentine's Day.

That's because scientists say watermelon has ingredients that deliver Viagra-like effects to the body's blood vessels and may even increase libido.

"The more we study watermelons, the more we realize just how amazing a fruit it is in providing natural enhancers to the human body," said Dr. Bhimu Patil, director of Texas A&M's Fruit and Vegetable Improvement Center in College Station.

"We've always known that watermelon is good for you, but the list of its very important healthful benefits grows longer with each study."

Beneficial ingredients in watermelon and other fruits and vegetables are known as phyto-nutrients, naturally occurring compounds that are bioactive, or able to react with the human body to trigger healthy reactions, Patil said.

In watermelons, these include lycopene, beta carotene and the rising star among its phyto-nutrients – citrulline – whose beneficial functions are now being unraveled. Among them is the ability to relax blood vessels, much like Viagra does.

Scientists know that when watermelon is consumed, citrulline is converted to arginine through certain enzymes. Arginine is an amino acid that works wonders on the heart and circulation system and maintains a good immune system, Patil said.

"The citrulline-arginine relationship helps heart health, the immune system and may prove to be very helpful for those who suffer from obesity and type 2 diabetes," said Patil. "Arginine boosts nitric oxide, which relaxes blood vessels, the same basic effect that Viagra has, to treat erectile dysfunction and maybe even prevent it."

While there are many psychological and physiological problems that can cause impotence, extra nitric oxide could help those who need increased blood flow, which would also help treat angina, high blood pressure and other cardiovascular problems.

"Watermelon may not be as organ specific as Viagra," Patil said, "but it's a great way to relax blood vessels without any drug side-effects."

The benefits of watermelon don't end there, he said. Arginine also helps the urea cycle by removing ammonia and other toxic compounds from our bodies.

Citrulline, the precursor to arginine, is found in higher concentrations in the rind of watermelons than the flesh. As the rind is not commonly eaten, two of Patil's fellow scientists, Drs. Steve King and Hae Jeen Bang, are working to breed new varieties with higher concentrations in the flesh.

In addition to the research by Texas A&M, watermelon's phyto-nutrients are being studied by the U.S. Department of Agriculture's Agricultural Research Service in Lane, Oklahoma.

As an added bonus, these studies have also shown that deep red varieties of watermelon have displaced the tomato as the lycopene king, Patil said. Almost 92 percent of watermelon is water, but the remaining 8 percent is loaded with lycopene, an anti-oxidant that protects the human heart, prostate and skin health.

"Lycopene, which is also found in red grapefruit, was historically thought to exist only in tomatoes," he said. "But now we know that it's found in higher concentrations in red watermelon varieties."

Lycopene, however, is fat-soluble, meaning that it needs certain fats in the blood for better absorption by the body, Patil said.

"Previous tests have shown that lycopene is much better absorbed from tomatoes when mixed in a salad with oily vegetables like avocado or spinach," Patil said. "That would also apply to the lycopene from watermelon, but I realize mixing watermelon with spinach or avocadoes is a very hard sell."

No studies have been conducted to determine the timing of the consumption of oily vegetables to improve lycopene absorption, he said.

"One final bit of advice for those Fourth of July watermelons you buy," Patil said. "They store much better uncut if you leave them at room temperature. Lycopene levels can be maintained even as it sits on your kitchen floor. But once you cut it, refrigerate. And enjoy."

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To Sing Like Shakira, Press "One" Now

TAU scientists have developed an electronic ear to judge and coach vibrato technique

Vibrato -- the pulsating change of pitch in a singer's voice -- is an important aspect of a singer's expression, used extensively by both classical opera singers and pop stars like Shakira. Usually, the quality of a vibrato can only be judged subjectively by voice experts.

Until now, that is. A research group from Tel Aviv University has successfully managed to train a computer to rate vibrato quality, and has created an application based on biofeedback to help singers improve their technique. Your computer can now be a singing coach.

The invention was recently showcased at an international competition in Istanbul, where it won first prize at the International Cultural and Academic Meeting of Engineering Students. Researcher Noam Amir, a senior lecturer from the Department of Communication Disorders at the Sackler Faculty of Medicine, Tel Aviv University, says the tool might not help record producers find the next great pop music sensation. But it could teach singers how to mimic Shakira's signature vibrato.

Good Singing Is Not Subjective

Vibrato is a musical effect than can be used when a musician sings or plays an instrument. It adds expression to a song and is created by a steady pulsating change of pitch, characterized by the amount of variation and the speed at which the pitch is varied. TAU's application can teach singers how to mimic the vibrato qualities most attractive to the human ear.

But mastering vibrato is no guarantee for an American Idol appearance. "Vibrato is just one aspect of a singer's impact," says Amir, an expert in the ways that emotions impact speech. "Singers need to arouse an emotional response, and that is a complicated task."

Music, By the Numbers

Three years ago, Amir and his colleagues decided that they would look for an objective, numerical assessment of vibrato quality. New vocal students usually don't have good control of their vibrato, explains Amir. "Their vibrato is erratic and hard to judge subjectively, and it's hard to find to a precise measure for this. We wanted to find a way to emulate a human expert in a computer program."

Amir's team input into their computer many recordings by students singing vibrato and had their vibrato judged by human teachers. Using hundreds of vocal students and expert judges, the team was able to use mathematical measurements to correlate vibrato styles to their quality as judged by the teachers.

The computer was then able to rate the vibrato quality of new voices on its own, producing ratings similar to those given by the expert vocal teachers. In effect, a machine had "learned" how to judge the quality of an individual singer's vibrato. The researchers then added a biofeedback loop and a monitor so that singers could see and augment their vibrato in real time.

An Escape from Call Centers

Other applications for this type of research, Amir says, could be in automated call centers, where callers communicate with computers. He hopes to be able to teach computers how to recognize a range of different emotions, such as anger and nervousness, so that a live receptionist can jump in when a caller becomes upset with the machine.

Amir's research focuses on how emotions are expressed in speech. He collaborates regularly with speech pathologists and in this particular study worked with Dr. Ofer Amir and Orit Michaely, also from the Sackler Faculty of Medicine.

The original research was published in the journal Biomedical Signal Processing and Control.

Life-extending protein can also have damaging effects on brain cells

Proteins widely believed to protect against aging can actually cause oxidative damage in mammalian brain cells, according to a new report in the July Cell Metabolism, a publication of Cell Press. The findings suggest that the proteins can have both proaging and protective functions, depending on the circumstances, the researchers said.

" Sirtuins are very important proteins," said Valter Longo of the University of Southern California, Los Angeles. "Overexpression can protect in some cases, and in other cases, it may do the opposite. It has to do with the fact that they do so many things."

Sirtuins, or Sir2 family proteins, are found in organisms from bacteria to humans. Sir2 controls aging and life span in yeast, the worm C. elegans, and Drosophila fruit flies, earlier studies have shown.

Studies have also implicated Sir2 in the life-extending effects of a calorie restricted diet in some, though not all, organisms. Notably, Longo's lab showed that lack of Sir2 in yeast further extended the life span of calorie-restricted cells.

SirT1, the mammalian version of yeast Sir2, controls numerous physiological processes including glucose metabolism, DNA repair, and cell death, the researchers added. In mammalian cells, SirT1 also controls several stress-response factors.

Now, the researchers show that cultured rat neurons treated with a SirT1 inhibitor more often survived treatment with oxidative stress-inducing chemicals. They further show evidence to explain the mechanism responsible for that effect.

They also found lower oxidative stress levels in the brains of mice without SirT1. However, those SirT1 knockout mice didn't live as long as normal mice do on either a normal or a calorie-restricted diet.

These results are consistent with the existence of a prooxidative stress role for mammalian SirT1 similar to that described for Sir2 in yeast but confirm that sirtuins can play both positive and negative roles, Longo said. Based on the new findings, Longo urges caution to those developing SirT1-boosting drugs intended for human consumption.

" [Such drugs] could have beneficial effects for certain diseases, but again, these proteins do a lot of things," he said. "I would say the idea that there is a conserved action of sirtuins to cause major life span extension—the foundations for that are weak or very weak. Until we have more data to show that chronic treatment to increase SirT1 activity does not do damage, I don't think it's a good idea."

The researchers include Ying Li, Neuroscience Program, University of Southern California, Los Angeles, CA, Andrus Gerontology Center and Department of Biological Sciences, University of Southern California, Los Angeles, CA; Wei Xu, Neuroscience Program, University of Southern California, Los Angeles, CA; Michael W. McBurney, Ottawa Health Research Institute, Department of Medicine, University of Ottawa, Ottawa, Ontario, Canada; and Valter D. Longo, Neuroscience Program, University of Southern California, Los Angeles, CA, Andrus Gerontology Center and Department of Biological Sciences, University of Southern California, Los Angeles, CA.

Cancer cells revert to normal at specific signal threshold, Stanford researchers find

STANFORD, Calif. - Cancer starts when key cellular signals run amok, driving uncontrolled cell growth. But scientists at the Stanford University School of Medicine report that lowering levels of one cancer signal under a specific threshold reverses this process in mice, returning tumor cells to their normal, healthy state. The finding could help target cancer chemotherapy to tumors while minimizing side effects for the body's healthy cells.

The researchers identified a precise threshold level of the signaling molecule Myc that determined the fate of tumor cells in a cancer of the immune system in mice. Above the threshold, high levels of Myc drove immune cells to grow too large and multiply uncontrollably. When the researchers lowered Myc levels below the threshold, the same cells shrank to normal size, stopped multiplying and began dying normally.

"This is a new concept," said Catherine Shachaf, PhD, an instructor in microbiology and immunology who shared lead authorship of the study with colleague Andrew Gentles, PhD, a research associate in radiology. Previous research demonstrated that turning Myc and other cancer signals all the way off can kill a tumor, but this is the first time scientists have demonstrated a specific midway point at which a cancer signal reverted to a healthy level, Shachaf said. The findings will be published in the July 1 issue of Cancer Research.

Identifying the threshold was important because Myc functions in both healthy and cancerous cells as a transcription factor, a protein signal that binds DNA to turn genes on or off. Excess Myc contributes to about 50 percent of human cancers, including malignancies of the immune system and lung.

But Myc is essential, at lower levels, for normal cell function. So, switching Myc all the way off is not an option for treating cancer.

"I wanted to figure out, if we had a drug to turn off Myc, how could we give it to people without hurting them?" said Dean Felsher, MD, PhD, associate professor of oncology and of pathology. Felsher and Sylvia

Plevritis, PhD, associate professor of radiology, are the study's senior authors and are both members of the Stanford Cancer Center.

In the past, scientists have shown that cancer signals such as Myc are "like light switches," Felsher said. "Now we know that, in some cases, you don't need to turn the light completely off."

"The real significance of this paper is that it demonstrates that there is a defined amount of Myc that switches the balance between normal cell growth and tumorigenesis," said Bill Tansey, PhD, a professor and expert on cancer-gene regulation at Cold Spring Harbor Laboratory in New York, who was not involved in the research. "The idea that this is a threshold is really not the way we were all thinking."

Using mice that were genetically engineered to develop Myc-driven tumors in response to a chemical in their drinking water, the researchers slowly lowered Myc from an elevated, cancer-causing level to the precise point at which tumor cells returned to normal. Near the threshold, they examined many aspects of cell metabolism to obtain a detailed picture of how the cancer cells changed as Myc dropped. They measured changes in gene activity, protein levels, protein activation inside the cells and the appearance of cell-labeling proteins on the exterior surface of the cells. The scientists wrote a new piece of computer software to help them see how these different types of data fit together into detailed metabolic pathways.

"At the Myc threshold, there is a big change: Programmed cell death becomes dominant over growth," said Gentles.

The threshold was characterized by both a return of normal controls on the cell's life cycle, which stopped inappropriate growth, and re-activation of the pathways that prompt normal cell death, Gentles said.

"We were able to experimentally prove that we can turn Myc off a little bit, or for a little time, and that's enough to have a profound effect on cancer," Felsher said.

The multidisciplinary research team that conducted the work included 14 scientists from seven different Stanford departments.

The study's results will be used to design future cancer treatments, the team said. At present, no drugs target Myc. Understanding the Myc threshold will make it easier to design new drugs that focus on Myc itself or target other key signals required to switch from tumor to healthy cells. Armed with a detailed profile of cellular changes near the Myc threshold, researchers now have a much better idea of where to look for new cancer treatments. "It allowed us to narrow down the hunt," Felsher said.

The research was supported by grants from the National Cancer Institute, the National Cancer Institute Integrative Cancer Biology Program, the Leukemia and Lymphoma Society, the Damon Runyon Foundation, the Burroughs Wellcome Fund, a Weiland Family Fellowship and a Flight Attendant Medical Research Institute Young Clinical Scientist Award.

Felsher and colleagues published a companion paper June 6 in Public Library of Science-Genetics examining bone cancer.

Spiritual effects of hallucinogens persist, Johns Hopkins researchers report

Related report gives safety guidelines for hallucinogen research

In a follow-up to research showing that psilocybin, a substance contained in "sacred mushrooms," produces substantial spiritual effects, a Johns Hopkins team reports that those beneficial effects appear to last more than a year.

Writing in the Journal of Psychopharmacology, the Johns Hopkins researchers note that most of the 36 volunteer subjects given psilocybin, under controlled conditions in a Hopkins study published in 2006, continued to say 14 months later that the experience increased their sense of well-being or life satisfaction.

"Most of the volunteers looked back on their experience up to 14 months later and rated it as the most, or one of the five most, personally meaningful and spiritually significant of their lives," says lead investigator Roland Griffiths, Ph.D., a professor in the Johns Hopkins departments of Psychiatry and Behavioral Sciences and Neuroscience.

In a related paper, also published in the Journal of Psychopharmacology, researchers offer recommendations for conducting this type of research.

The guidelines caution against giving hallucinogens to people at risk for psychosis or certain other serious mental disorders. Detailed guidance is also provided for preparing participants and providing psychological support during and after the hallucinogen experience. These "best practices" contribute both to safety and to the standardization called for in human research.

"With appropriately screened and prepared individuals, under supportive conditions and with adequate supervision, hallucinogens can be given with a level of safety that compares favorably with many human research and medical procedures," says that paper's lead author, Mathew W. Johnson, Ph.D., a psychopharmacologist and instructor in the Johns Hopkins Department of Psychiatry and Behavioral Sciences.

The two reports follow a 2006 study published in another journal, Psychopharmacology, in which 60 percent of a group of 36 healthy, well-educated volunteers with active spiritual lives reported having a "full mystical experience" after taking psilocybin. *{See http://www.hopkinsmedicine.org/Press_releases/2006/07_11_06.html}*

Psilocybin, a plant alkaloid, exerts its influence on some of the same brain receptors that respond to the neurotransmitter serotonin. Mushrooms containing psilocybin have been used in some cultures for hundreds of years or more for religious, divinatory and healing purposes.

Fourteen months later, Griffiths re-administered the questionnaires used in the first study -- along with a specially designed set of follow up questions -- to all 36 subjects. Results showed that about the same proportion of the volunteers ranked their experience in the study as the single most, or one of the five most, personally meaningful or spiritually significant events of their lives and regarded it as having increased their sense of well-being or life satisfaction.

"This is a truly remarkable finding," Griffiths says. "Rarely in psychological research do we see such persistently positive reports from a single event in the laboratory. This gives credence to the claims that the mystical-type experiences some people have during hallucinogen sessions may help patients suffering from cancer-related anxiety or depression and may serve as a potential treatment for drug dependence. We're eager to move ahead with that research."

Griffiths also notes that, "while some of our subjects reported strong fear or anxiety for a portion of their day-long psilocybin sessions, none reported any lingering harmful effects, and we didn't observe any clinical evidence of harm."

The research team cautions that if hallucinogens are used in less well supervised settings, the possible fear or anxiety responses could lead to harmful behaviors.

These studies were funded by grants from NIDA, the Council on Spiritual Practices, and the Heffter Research Institute. Additional researchers who contributed to this work include Matthew W. Johnson, Ph.D. and Una D. McCann, M.D. of the Department of Psychiatry and Behavioral Sciences, Johns Hopkins University School of Medicine; psychologist William A. Richards of the Johns Hopkins Bayview Medical Center; and Robert Jesse of the Council on Spiritual Practices, San Francisco.

Designer diet for prostate cancer

Eating one or more portions of broccoli every week can reduce the risk of prostate cancer, and the risk of localised cancer becoming more aggressive.

For the first time, a research group at the Institute of Food Research led by Professor Richard Mithen has provided an explanation of how eating broccoli might reduce cancer risk based upon studies in men, as opposed to trying to extrapolate from animal models. Prostate cancer is the most common non-skin cancer for males in western countries. The research has provided an insight into why eating broccoli can help men stay healthy.

For the study, published in the online, open-access journal PLoS ONE on July 2, men who were at risk of developing prostate cancer ate either 400g of broccoli or 400g of peas per week in addition to their normal diet over 12 months. Tissue samples were taken from their prostate gland before the start of the trial and after 6 and 12 months, and the expression of every gene measured using Affymetrix microarray technology.

It was found that there were more changes in gene expression in men who were on the broccoli-rich diet than on the pea diet, and these changes may be associated with the reduction in the risk of developing cancer, that has been reported in epidemiological studies.

Previous studies have suggested that the fifty percent of the population who have a GSTM1 gene gain more benefit from eating broccoli than those who lack this gene. The study showed that the presence of the GSTM1 gene had a profound effect on the changes in gene expression caused by eating broccoli.

This study fills the gap between observational studies and studies with cell and animal models. While observational studies have shown that diets rich in cruciferous vegetables may reduce the risk of prostate cancer and other chronic disease, they do not provide an explanation of how this occurs. Evidence from animal and cell models has sought to provide an explanation, but these studies are usually based on high doses that would not normally be experienced as part of the diet.

The results of the study suggested that relatively low amounts of cruciferous vegetables in the diet – a few portions per week – can have large effects on gene expression by changing cell signalling pathways. These signalling pathways are the routes by which information is transmitted through a molecular cascade which amplifies the signal to the nucleus of the cell where gene expression occurs.

The Norwich-based team are currently planning a larger study with men with localised prostate cancer, and will compare the activity of standard broccoli with the special variety of high glucosinolate broccoli used in the current study.

Designer studies for health promotion

"Other fruits and vegetables have been shown to also reduce the risk of prostate cancer and are likely to act through other mechanisms," says Professor Mithen.

"Once we understand these, we can provide much better dietary advice in which specific combinations of fruit and vegetable are likely to be particularly beneficial. Until then, eating two or three portions of cruciferous vegetable per week, and maybe a few more if you lack the GSTM1 gene, should be encouraged."

Notes for editors:

Funding: The work was supported by the Biotechnology and Biological Sciences Research Council (BBSRC).

Cruciferous vegetables include broccoli, Brussels sprouts, cauliflower, cabbage, rocket, watercress, garden cress, kale, bok choy, radish, horseradish and wasabi.

The broccoli used in this study is a high glucosinolate variety. The variety was developed at the John Innes Centre in Norwich, and then licensed to Seminis Inc for commercialisation by Plant Bioscience Ltd.

Resuscitation technique after brain injury may do more harm than good, researchers find

DALLAS — July 1, 2008 — The current standard practice of giving infants and children 100 percent oxygen to prevent brain damage caused by oxygen deprivation may actually inflict additional harm, researchers at UT Southwestern Medical Center have found.

Brain damage caused by oxygen deprivation, known as hypoxic-ischemic brain injury, is one of the most common causes of death and long-term neurological damage among infants and children. This can happen during birth trauma, near drowning and other crises.

The UT Southwestern researchers found that mice treated with less than a minute of 100 percent oxygen after a hypoxic-ischemic brain injury suffered far greater rates of brain-cell death and coordination problems similar to cerebral palsy than those allowed to recover with room air.

"This study suggests 100 percent oxygen resuscitation may further damage an already compromised brain," said Dr. Steven Kernie, associate professor of pediatrics and developmental biology and senior author of the study, which appears in the July issue of the Journal of Cerebral Blood Flow & Metabolism.

Most of the damage involved cells that create myelin, a fatty substance that insulates nerve cells and allows them to transmit electrical signals quickly and efficiently. Infants have much less myelin than adults; as myelin develops in children they become more coordinated. Areas of the brain with dense areas of myelin appear white, hence the term "white matter."

"Patients with white-matter injuries develop defects that often result in cerebral palsy and motor deficits," Dr. Kernie said.

Myelin comes from cells called glial cells, or glia, which reach out and wrap part of their fatty membranes around the extensions of nerve cells that pass electrical signals. The brain creates and renews its population of glial cells from a pool of immature cells that can develop into mature glia.

In their study, the researchers briefly deprived mice of oxygen, then gave them either 100 percent oxygen or room air, which contains about 21 percent oxygen, 78 percent nitrogen and 1 percent other gases.

After 72 hours, mice given 100 percent oxygen fared worse than those given room air. For example, they experienced a more disrupted pattern of myelination and developed a motor deficit that mimicked cerebral palsy.

The population of immature glial cells also diminished, suggesting that the animals would have trouble replacing the myelin in the long term.

"We wanted to determine whether recovery in 100 percent oxygen after this sort of brain injury would exacerbate neuronal injury and impair functional recovery, and in these animals, it did impair recovery," Dr. Kernie said. "Our research shows even brief exposure to 100 percent oxygen during resuscitation actually worsens white-matter injuries."

Dr. Kernie said adding pure oxygen to the damaged brain increases a process called oxidative stress, caused by the formation of highly reactive molecules. The researchers found, however, that administering an antioxidant, which halts the harmful oxidation process, reversed the damage in the mice given 100 percent oxygen.

"Further research is needed to determine the best possible concentration of oxygen to use for optimal recovery and to limit secondary brain injury," Dr. Kernie said. "Research is now being done to determine the best way to monitor this sort of brain damage in humans so we can understand how it correlates to the mouse models. There are many emerging noninvasive technologies that can monitor the brain."

Other UT Southwestern researchers involved in the study were Dr. Joshua Koch, a pediatric clinical care fellow and lead author of the study; Darryl Miles, a pediatric clinical instructor; Jennifer Gilley, a student research assistant in pediatrics and developmental biology; and Dr. Cui-Pang Yang, a postdoctoral researcher in pediatrics and developmental biology.

'Hibernation-on-demand' drug significantly improves survival after extreme blood loss

Findings in rats show promise for victims of lethal hemorrhage

SEATTLE – For the first time, researchers have demonstrated that the administration of minute amounts of inhaled or intravenous hydrogen sulfide, or H2S – the molecule that gives rotten eggs their sulfurous stench – significantly improves survival from extreme blood loss in rats.

Cell biologist Mark B. Roth, Ph.D., and colleagues in the Basic Sciences Division of Fred Hutchinson Cancer Research Center, in collaboration with surgeon Robert K. Winn, Ph.D., and colleagues at UW Medicine's Harborview Medical Center, report their findings online ahead of print in The Journal of Trauma Injury, Infection, and Critical Care. The article is slated for the July print issue, which comes out on July 10.

The researchers successfully used H2S to induce a state of reversible metabolic hibernation as a way to reduce death from insufficient blood supply to organs and tissues in a rat model of lethal hemorrhage. (Federal regulations mandate the use of such animal models in preclinical research to test the safety and effectiveness of various procedures and treatments before they can be tested in humans.)

They found that 75 percent of rats (18 of 24) given inhaled hydrogen sulfide and 67 percent of rats (eight of 12) given intravenous hydrogen sulfide survived at least two weeks – the duration of the monitoring period – after losing more than half of their blood for an extended period. In contrast, long-term survival rates for the untreated rats in the two control groups were 23 percent (three of 13) and 14 percent (one of seven), respectively.

"Our goal is to develop life-saving treatment for critically ill people suffering from acute, sustained blood loss, such as in a car accident or on the battlefield," said senior author Roth. "These findings have obvious implications for the military, but they also have tremendous implications for the civilian population."

The U.S. Defense Advanced Research Projects Agency and the U.S. Defense Services Office funded the research. The ultimate goal: designing self-injectable hydrogen-sulfide kits that critically injured soldiers could use in the field to temporarily dim their metabolism and reduce their oxygen demand. This would help "buy time" until they could get medical attention.

"The military feels that if a soldier can be kept alive for at least three hours, that would allow time for the situation to be stabilized and the scene of the incident secured enough to allow evacuation of that soldier to an area where he or she can get medical attention," Roth said.

Roth's study, which attempted to mimic a similar scenario, involved 56 rats, each of which underwent controlled hemorrhage to remove 60 percent of their blood for three hours before re-infusion with Lactated Ringer's solution to replace lost blood volume.

The rats were divided into two groups. In the first group, 24 rats were put into a controlled atmosphere of room air laced with 300 parts per million H2S while 13 served as controls. The H2S was administered about 20 minutes after initiation of blood removal and was supplied for about 20 minutes, until the end of the bleed. In the second group, 12 rats received a single intravenous dose of sulfide solution about 20 minutes after the initiation of blood removal while seven served as controls.

In both test groups, the rats maintained a reduced yet stable level of carbon-dioxide production, a surrogate measure of metabolism. Once H2S was removed, metabolic rates returned to normal. In contrast, the untreated animals steadily grew metabolically weaker from blood loss until the point of death.

Functional and behavioral testing among the long-term survivors (those that lived more than two weeks after hemorrhage) showed no observable defects. In fact, the rodents that were bred produced normal-sized litters of healthy pups.

How does hydrogen-sulfide treatment prevent death from profound and sustained blood loss? One possibility is that in reducing metabolism, H2S also reduces oxygen demand, which allows crucial neurons in the hippocampus, the part of the brain that controls autonomic functions such as breathing and heartbeat, to withstand low oxygen levels due to hemorrhage.

Another mechanism may be that hydrogen sulfide, which is naturally present in the blood, is lost during hemorrhage and must be replaced to maintain life processes.

In April 2005 Roth and colleagues made headlines worldwide when they reported, in the journal Science, the first use of H2S to induce a state of reversible hibernation in mice. Roth's latest research represents the next step in demonstrating hydrogen sulfide's potential to treat ischemic injuries caused by conditions such as severe blood loss, hypothermia, cardiac arrest and stroke.

Counting monkeys tick off yet another 'human' ability

* 11:03 01 July 2008
* NewScientist.com news service
*** Ewen Callaway**

At this rate a monkey might prove the Riemann hypothesis. Rhesus macaques have been shown to possess yet another numerical talent once thought unique to humans – they can simultaneously count audible beeps and dots on a computer screen.

Their ability to comprehend numbers not as just discrete images or sounds, but as abstract representations that can be combined suggests that such maths skills aren't unique to humans, says Kerry Jordan, a psychologist at Utah State University, Logan, US, who led the new study.

This sort of evidence "shows that [animals] have these precursors to math very early on in the evolutionary line and early on in development," she says.

Dotty tasks

Jordan and colleague Elizabeth Brannon, of Duke University in Durham, North Carolina, US, trained two eight-year-old female macaques to equate beeps to dots on a computer screen. So if a monkey heard seven beeps, it knew to tap a square on the screen displaying seven dots.

Next, the researchers tested the monkeys' training in adding dots and beeps together.

The animals were presented dots of different sizes flash onto a screen. At the same time they heard a series of short tones.

To determine if the monkeys could combine the two, Jordan and Brannon showed the animals a screen with two numerical choices, represented as dots – one the correct sum, one incorrect.

Both monkeys did better than 50:50 – one added the sights and sounds correctly 72% of the time, the other 66% of the time.

Both monkeys tended to make mistakes when the right and wrong answers were numerically similar. For instance, if the choices were one and eight, the animals rarely got it wrong. But they found it harder to choose between, say, five and six.

Wild skills

People make the same kind of errors when making snap numerical judgements, such counting the number of people in a crowd, says Jordan, which is further evidence that our abstract maths skills aren't unique.

The monkey's ability to add numbers seen and heard together makes sense in the wild, says Jordan.

"If you have an animal trying to make a decision to defend its territory, it's going to want know how many other animals it has to deal with," she says. It would do this by combining information on how many animals it could see with how many it could hear.

Irene Pepperberg, a psychologist at Harvard University in Cambridge, Massachusetts, who trained a parrot named Alex to add small sums, says the paper confirms observations in the wild.

Flycatchers, for instance, seem to communicate their mood to other birds using a numerical combination of song and wing motions. The more wing flicks and songs, the more likely it is to attack another bird, she says.

Journal reference: Cognition, DOI: 10.1016/j.cognition.2008.05.006

Well

Diabetes: Underrated, Insidious and Deadly

By TARA PARKER-POPE

In a set of recent focus groups, participants were asked to rank the severity of various health problems, including cancer, heart disease and diabetes.

On a scale of 1 to 10, cancer and heart disease consistently ranked as 9s and 10s. But diabetes scored only 4s and 5s.

"The general consensus seems to be, 'There's medication,' 'Look how good people look with diabetes' or 'I've never heard of anybody dying of diabetes,' " said Larry Hausner, chief executive of the American Diabetes Association, which held the focus groups. "There was so little understanding about everything that dealt with diabetes."

But diabetes is anything but minor. It wreaks havoc on the entire body, affecting everything from hearing and vision to sexual function, mental health and sleep. It is the leading cause of blindness, amputations and kidney failure, and it can triple the risk for heart attack and stroke.

Stuart Bradford

“It is a disease that does have the ability to eat you alive,” said Dr. John B. Buse, a professor at the University of North Carolina School of Medicine who is the diabetes association’s president for medicine and science. “It can be just awful — it’s almost unimaginable how bad it can be.”

Diabetes results when the body cannot use blood sugar as energy, either because it has too little insulin or because it cannot use insulin. Type 2 diabetes, which accounts for 90 to 95 percent of cases, typically develops later in life and is associated with obesity and lack of exercise. Type 1 diabetes, which is often diagnosed in children, occurs when the immune system mistakenly destroys cells that make the insulin.

The disconnect between perception and reality is particularly worrisome at a time when national diabetes rates are surging. Just last week, the Centers for Disease Control and Prevention announced that the number of Americans with diabetes had grown to about 24 million, or 8 percent of the population. Almost 25 percent of those aged 60 and older had diabetes in 2007. And the C.D.C. estimates that 57 million people have abnormal blood sugar levels that qualify as pre-diabetes.

To be sure, diabetes is treatable, and an array of new medications and monitoring tools have dramatically improved the quality of care. But keeping the illness in check requires constant vigilance and expensive care, along with lifestyle changes like losing weight, exercising regularly and watching your carbohydrates.

Dr. Buse says patients who are focused on their disease and who have access to regular medical care have a good chance of living out a normal life span without developing a diabetes-related disability.

But some patients say they are too busy to take better care of themselves, and many low-income patients can’t afford regular care. Even people with health insurance struggle to keep up with the co-payments for frequent doctor visits and multiple medications.

And to make matters worse, diabetes is associated with numerous other health problems. Last week, for example, The Journal of the American Medical Association reported that people with depression were at higher risk for Type 2 diabetes, and vice versa.

That is not surprising: according to data published last year in the journal Diabetes Care, depression tends to interfere with a patient’s self-care, which requires glucose monitoring, medications, dietary changes and exercise.

Ultimately, diabetes can take a toll from head to toe. In the brain, it raises the risk not only for depression but also for sleep problems and stroke. It endangers vision and dental health. This month, The Annals of Internal Medicine is reporting that the disease more than doubles the risk of hearing loss.

Moving down the body, diabetes can lead to liver and kidney disease, along with serious gastrointestinal complications like paralysis of the stomach and loss of bowel control. Last year the journal Diabetes Care reported that in a sample of nearly 3,000 patients with diabetes, 70 percent had nonalcohol fatty liver disease.

Poor circulation and a loss of feeling in the extremities, called neuropathy, can lead to severe ulcers and infections; each year in the United States, there are about 86,000 diabetes-related amputations.

Diabetes can also take a toll on relationships. By some estimates, 50 percent to 80 percent of men with diabetes suffer from erectile dysfunction. Experts say women with diabetes often lose their libidos or suffer from vaginal dryness.

The challenge for doctors is to convince patients that diabetes is a major health threat. For years, the message from the American Diabetes Association has been one of reassurance that the disease is treatable. Now, beginning in 2009, the association plans to reframe its message to better communicate the seriousness of the disease.

“Our communication strategy is going to be that diabetes has deadly consequences, and that the A.D.A. is here to change the future of diabetes,” said Mr. Hausner, a former executive with the Leukemia and Lymphoma Society who came to the association 10 months ago. “It’s the word ‘deadly’ that was the potentially controversial word for the organization. In the past, people said, ‘We don’t want to get anybody scared.’ ”

The new strategy is not a scare tactic, he added. Prevention and hope will still be part of the message.

“It’s not that we don’t want people to have hope,” he said. “We want people to understand this is serious.”

Cases

Her Skin Erupted, and the Detective Work Began

By INGFEI CHEN

For years, since her 20s, my sister lived with dry, irritated little brown splotches of eczema on her neck, a pesky annoyance. But two years ago, something changed. Angry red rashes began marching across her body in an onslaught of itchy misery she feared would never end.

It was an odd case from the start. Chronic eczema is a complex disorder that involves an overwrought immune system and, often, a defective gene that leaves the skin barrier leaky, dry and easily irritated. Usually a

childhood affliction, it seldom strikes so severely for the first time in adults. Then again, we are talking about my sister — a 40-something Taiwanese-American who is inexplicably allergic to Chinese food.

In November 2006, what began as a few small, puffy red spots on her shins escalated into large, unsightly rashes. On Thanksgiving eve, they were pocked with pimplelike bumps and oozed pus from crusted cracks. Alarmingly, her lower legs had swollen to the ankles. I drove her to an urgent-care center, where a doctor prescribed antibiotics.

Over the weekend, itchy spots broke out on her arms, back and belly. The tops of her feet had no rashes, but at bedtime, she recalled, they grew "insanely itchy." Sleep was impossible.

On Monday, she saw a dermatologist, who said a bacterial infection — Staphylococcus aureus, a lab test revealed — had accelerated a full-body eczema outbreak. It was bizarre enough that three other doctors crowded into the room to ogle her rash under a magnifying lamp.

Her dermatologist gave her a shot of potent, anti-inflammatory cortisone and prescribed a daily corticosteroid ointment. Among other advice, he warned against too much showering or bathing because that dries the skin, irritating it; and the more so with hot water. Lukewarm water was best.

My sister cut back to showering every other day and generously moisturized with petroleum jelly. A prescription antihistamine aided her sleep, but left her zombielike at work.

The skin symptoms retreated, but only temporarily. My sister's sensitive hide was quick to take umbrage — at dry air, warm temperatures, the roughness of denim, even stressful thoughts. She fell into a recurring cycle of flare-ups, and got a second cortisone shot. The doctor told her she might have to cope with the problem for the rest of her life.

"I couldn't believe it was happening," she said. "It was disturbing."

The eczema was leaving big, leathery brown scars. "You look like a giraffe," I blurted one day. She glared. And, with despair, banished short sleeves and skirts to the back of her closet.

In summer 2007, a friend mentioned that he had received a respite from his own persistent eczema by taking antibiotics, even though his lesions were uninfected. That was surprising. We assumed the antibiotics my sister took in November had eliminated her staph problem, but could bacteria still be fueling her rashes?

In August, she sought a second opinion from another dermatologist. Along with a higher-potency topical corticosteroid, that doctor agreed to prescribe antibiotic pills, along with a bacteria-killing ointment called mupirocin — which Sis was to dab up her nose. What's more, she was supposed to regularly dunk herself in a Clorox bath.

Yes: household bleach. It turns out that scientists have long known that 90 percent of chronic eczema sufferers, unlike healthy people, carry Staph aureus on their skin. The nose, too, is a reservoir of the microbe, which touches off inflammation-stoking mechanisms that make rashes worse, according to Dr. Amy S. Paller, a pediatric dermatologist at Northwestern University.

Concerned over the rise of drug-resistant bacteria, dermatologists typically resort to antibiotics only when eczema lesions show active infection. But a cheap alternative for reducing skin microbes is a gentle bleach bath (half a cup of Clorox in a full bathtub). Dr. Paller recently completed the first study to rigorously test bleach baths against eczema, and she said the results, not yet published, were promising.

Focusing on the staph, it turned out, made all the difference. Rather than Clorox baths, Sis preferred wiping her lesions every few days with a compress soaked with diluted bleach. Even before she finished 14 days of antibiotics — which also have some anti-inflammatory effects — her lesions calmed down markedly. By the end of August, for the first time in a year, there were no flared patches on her legs.

Still, concerned she would relapse, I decided to use my reporter's skills to learn more about eczema. Two interesting insights emerged.

In September, at an esoteric meeting called the Fourth International Workshop for the Study of Itch, I heard that, curiously, pain can suppress itching, because the neural circuits for the two sensations appear to overlap. Just as scratching, a form of pain, relieves itching, studies find that "noxious" stimuli — heat or electric shocks — can do the same. Conversely, cancer patients taking epidural injections of morphine can develop terrible itches.

Two months later, I witnessed this inverse phenomenon in my sister. With the fall weather, her eczema resurged. Then she injured her neck, and excruciating pain spread to her shoulders. Within days, the rashes ebbed remarkably.

Coincidence? Maybe. Anyway, neither ethicists nor my parents would have approved pulling out my sister's fingernails to cure her itch. After her neck improved, the eczema reared up again in December. She was reluctant to take antibiotics again, though.

But in February another useful (and pain-free) solution surfaced. While writing the Health Guide entry on eczema for nytimes.com/health, I discovered that the advice my sister received on bathing — that too much was bad — was outdated.

"That's an old misconception," Dr. Jon Hanifin, a dermatologist at Oregon Health and Science University, told me. For severe eczema, he advises 20-minute lukewarm baths twice a day for a week.

Eczema patients need frequent bathing, he said — not just to clean off bacteria but to rehydrate their parched epidermis. True, evaporation after a bath tends to dry out the skin. But so long as you lock in moisture by immediately applying topical corticosteroid or skin cream, "it's much better for you," Dr. Hanifin said. Steroids penetrate more effectively into hydrated skin.

Dr. Hanifin e-mailed me a handout on skin care, which I gave my sister. She returned to daily bathing and added tub baths, with Epsom salt, back to her routine. But she indulged in hot — not lukewarm — water. "I don't like sitting there and shivering," she said. She also increased the bleach compresses to every day.

Today, her arms and torso are mostly clear of eczema; the giraffe scars are fading. Her legs are much better too, though some problem spots linger. Although she expects more flare-ups, she finally knows how to control them. "Being more informed," she said, "takes a bit of the stress away."

It remains a mystery why eczema erupted so fiercely into my sister's life. But the lesson we took from it is tried and true: making the effort to understand a medical condition and the details of how best to treat it, really pays off. So does persistence.

"You just have to try everything, and you can't expect a doctor to know precisely what's going to happen to you," my sister said. A physician's training is based on caring for an average patient, "and you may not be an average person."

Certainly, my sister is not. *Ingfei Chen is a science writer in the San Francisco Bay Area.*

Worms do calculus to find meals or avoid unpleasantness

Oregon researchers identify computer-like mechanism that drives neuron expression for taste and smell

Thanks to salt and hot chili peppers, researchers have found a calculus-computing center that tells a roundworm to go forward toward dinner or turn to broaden the search. It's a computational mechanism, they say, that is similar to what drives hungry college students to a pizza.

These behavior-driving calculations, according to a paper published in the July 3 issue of the journal Nature, are done "in a tiny, specialized computer inside a primitive roundworm," says principal investigator Shawn Lockery, a University of Oregon biologist and member of the UO Institute of Neuroscience.

In their paper, the researchers documented how two related, closely located chemosensory neurons, acting in tandem, regulate behavior. The left neuron controls an on switch, while the opposing right one an off switch. These sister neurons are situated much like the two nostrils or two eyes of mammals. Together these neurons are known as ASE for antagonistic sensory cues.

It's possible, Lockery said, that the discovery someday could help research aimed at treating at least some of the 200,000 people in the United States who annually seek medical treatment, according to records of the National Institutes of Health, for problems involving taste and smell.

A spike in salt concentration in ASEL (left neuron) activates expression that leads a worm to proceed in a straight line. A dip in salt levels in ASER (right neuron)...

Click here for more information.

"This computer does some nice calculus, differentiating the rate of change of the strength of various tastes," Lockery said. "The worm uses this information to find food and to avoid poisons."

Lockery and colleagues predicted the existence of a derivative-crunching mechanism in the Journal of Neuroscience in 1999 based on findings that nematodes change directions based on taste and smell.

"In effect, they have two nostrils or two tongues but they are so close together that it is really like having one nostril or one tongue, and yet they find their way around quite effectively," Lockery said. "We knew from behavioral experiments that nematodes were doing the same thing that humans were doing, but only from the view of behavioral responses. We didn't know what was going on in the brain."

To get there, Lockery and colleagues used new imaging and molecular tools, along with some genetic engineering of their worms.

In one experiment, these chemosensory neurons carried a fluorescent protein that changed color based on neuronal activity. In another experiment, the neurons carried receptor proteins that recognize capsaicin, the active component in chili peppers.

Researchers found that when concentrations of salt were high, fluorescent proteins change from blue to yellow, showing that the left neuron (ASEL) was active as the worms continued forward movement. When salt levels were reduced, the right neuron (ASER) activated but generated a different behavior; the worms began a turning, or searching, motion.

"At this point, we wanted to know if these neurons really are controlling behavior. If ASEL really signals that things are getting better, then, if you could artificially activate ASEL the animals ought to go straight like a human going directly toward the pizza," Lockery said. "Conversely, if you activate the ASER the animals ought to turn to find their goal."

Such was the case, according to the capsaicin-receptor experiment. When the pepper ingredient was spread on turning worms with receptor proteins in the left neuron, they straightened their motion. Likewise, capsaicin applied to worms with the receptors in their right neurons caused them to change from turning motion to forward crawling.

"We have discovered a tiny, specialized computer inside a primitive round worm," Lockery said. "The computer calculates the rate of change of the strengths, or concentrations, of various tastes. The worm uses this information to find food and to avoid poisons."

Evidence for such on and off switching cells in other chemosensory networks of mammals, he added, "There are strong indications that a similar device exists in the human nervous system."

Five co-authors with Lockery on the Nature paper were: Hiroshi Suzuki and William R. Schafer, both of the University of California, San Diego; Tod R. Thiele and Serge Faumont, both of the UO Institute of Neuroscience; and Marina Ezcurra of the Medical Research Council's Laboratory of Molecular Biology in Cambridge, UK. Suzuki and Schafer currently are with the Center for Research in Neurodegenerative Diseases at the University of Toronto.

The National Institutes of Health, National Science Foundation and the United Kingdom's Biotechnology and Biological Sciences Research Council's Human Frontier Science Program funded the research.

Discovery explains how cold sore virus hides during inactive phase

DURHAM, N.C. – Now that Duke University Medical Center scientists have figured out how the virus that causes cold sores hides out, they may have a way to wake it up and kill it.

Cold sores, painful, unsightly blemishes around the mouth, have so far evaded a cure or even prevention. They're known to be caused by the herpes simplex virus 1 (HSV1), which lies dormant in the trigeminal nerve of the face until triggered to reawaken by excessive sunlight, fever, or other stresses.

"We have provided a molecular understanding of how HSV1 hides and then switches back and forth between the latent (hidden) and active phases," said Bryan Cullen, Duke professor of molecular genetics and microbiology.

His group's findings, published in Nature, also provide a framework for studying other latent viruses, such as the chicken pox virus, which can return later in life as a case of shingles, and herpes simplex 2 virus, a genitally transmitted virus that also causes painful sores, Cullen said.

Most of the time, HSV1 lives quietly for years, out of reach of any therapy we have against it. It does not replicate itself during this time and only produces one molecular product, called latency associated transcript RNA or LAT RNA.

"It has always been a mystery what this product, LAT RNA, does," Cullen said. "Usually viral RNAs exist to make proteins that are of use to the virus, but this LAT RNA is extremely unstable and does not make any proteins."

In studies of mice, the team showed that the LAT RNA is processed into smaller strands, called microRNAs, that block production of the proteins that make the virus turn on active replication. As long as the supply of microRNAs is sufficient, the virus stays dormant.

After a larger stress, however, the virus starts making more messenger RNA than the supply of microRNAs can block, and protein manufacturing begins again. This tips the balance, and the virus ultimately makes proteins that begin active viral replication.

The new supply of viruses then travels back down the trigeminal nerve, to the site of the initial infection at the mouth. A cold sore always erupts in the same place and is the source of viruses that might infect another person, either from direct contact, or sharing eating utensils or towels, Cullen said.

The approach to curing this nuisance would be a combination therapy, Cullen said. "Inactive virus is completely untouchable by any treatment we have. Unless you activate the virus, you can't kill it," he said.

Cullen and his team are testing a new drug designed to very precisely bind to the microRNAs that keep the virus dormant. If it works, the virus would become activated and start replicating.

Once the virus is active, a patient would then take acyclovir, a drug that effectively kills replicating HSV1.

"In principle, you could activate and then kill all of the virus in a patient," Cullen said. "This would completely cure a person, and you would never get another cold sore."

He and the team are working with drug development companies in animal trials to begin to answer questions about how to deliver this drug most effectively.

Co-authors included Jennifer Lin Umbach, Ph.D., and Heather W. Karnowski, B.S., of the Duke Department of Molecular Genetics and Microbiology and Center for Virology, and Martha F. Kramer, Igor Jurak, and Prof. Donald M. Coen of the Department of Biological Chemistry and Molecular Pharmacology at Harvard Medical School. This work was supported by two NIH grants.

New antibiotic beats superbugs at their own game

The problem with antibiotics is that, eventually, bacteria outsmart them and become resistant. But by targeting the gene that confers such resistance, a new drug may be able to finally outwit them. Rockefeller University scientists tested the new drug, called Ceftobiprole, against some of the deadliest strains of multidrug-resistant Staphylococcus aureus (MRSA) bacteria, which are responsible for the great majority of staphylococcal infections worldwide, both in hospitals and in the community.

The research, to be published in the August 2008 issue of the journal Antimicrobial Agents and Chemotherapy and available online now, looked at how well Ceftobiprole worked against bacterial clones that had already developed resistance to other drugs. In every case, Ceftobiprole won. "It just knocked out the cells 100 percent," says the study's lead investigator, Alexander Tomasz, head of the Laboratory of Microbiology at Rockefeller.

Previous research had already shown that -- in general -- Ceftobiprole was highly effective against most clinical isolates of S. aureus. "Instead, we looked more carefully at the highly resistant cells that already occur in such clinical isolates at very low frequency -- maybe in one bacterium in every 1,000," says Tomasz. Ceftobiprole was able to kill these resistant cells.

Never before has an antibiotic been tested this way. "In the history of antibiotic development, an antibiotic arrives on the scene, and sooner or later resistant bacteria emerge," Tomasz says. "We sought to test in advance which would win this particular chess game: the new drug, or the bacteria that now cause human deaths."

In an ominous new "move" in this chess game, S. aureus strains with resistance to vancomycin (VRSA), a different class of antibiotics, also began to appear in hospitals in the United States. Ceftobiprole was also able to kill these new resistant VRSA strains.

The drug is effective because the chemists who developed Ceftobiprole managed to outwit the bacteria at their own game, Tomasz says. The broad-spectrum antibiotic was discovered by Basilea Pharmaceuticals, based in Basel, Switzerland, and is being developed in the U.S. and worldwide by Johnson & Johnson. The research was supported by Johnson & Johnson along with a grant from the U.S. Public Health Service.

Did newborn Earth harbour life?

* 18:03 02 July 2008

* NewScientist.com news service

*** Rachel Courtland**

Life on Earth might have emerged about 750 million years earlier than previously thought, new research suggests.

Researchers have found unusually light isotopes of carbon, a common indicator of life, in the Earth's oldest mineral deposit, found in the Jack Hills in Western Australia. The carbon dates to more than 4.25 billion years ago, a time known as the Hadean period.

Life is largely considered to have emerged around 3.5 billion years ago, after a violent period known as the Late Heavy Bombardment, in which a large amount of space debris walloped and may have sterilised the Earth.

But the Jack Hills find suggests life might have existed well before that time, although researchers caution it is too early to draw a definite conclusion.

"We now have an indication that it might be life," says mineralogist Thorsten Geisler of the Institute for Mineralogy at the University of Münster in Germany.

Geisler and colleagues dated samples from the Jack Hills area by measuring the abundance of radioactive elements in zircon deposits. They then analysed the concentration of carbon-13 and carbon-12 found in small pieces of diamond and graphite trapped within the zircon.

Organic material

They found the ratio of carbon-12, a lighter isotope of carbon, to carbon-13 was unusually high. Light carbon suggests the presence of organic material.

But it is too early to say for certain whether the carbon might indicate life. "We can't say now that we have unambiguous evidence of life before the Late Heavy Bombardment," Geisler told New Scientist.

That's because certain non-biological chemical reactions can also create light carbon, although the ratio is so skewed towards the lighter isotope that these reactions can't easily account for it.

A reservoir of light carbon might also indicate that simple organic compounds might have existed on Earth, priming the environment for the later emergence of life.

"When I see that, that's really good news, because we need a reservoir of reduced carbon compound to set the stage for the origin of life," says Jeffrey Bada, a chemist at the Scripps Institution of Oceanography in La Jolla, California, US. *Journal reference: Nature (vol 454, p 92)*

Exploding Asteroid Theory Strengthened by New Evidence Located in Ohio, Indiana

Was the course of life on the planet altered 12,900 years ago by a giant comet exploding over Canada? New evidence found by UC Assistant Professor of Anthropology Ken Tankersley and colleagues suggests the answer is affirmative.

Date: 7/2/2008

By: **Carey Hoffman**

UC ingot Geological evidence found in Ohio and Indiana in recent weeks is strengthening the case to attribute what happened 12,900 years ago in North America -- when the end of the last Ice Age unexpectedly turned into a phase of extinction for animals and humans – to a cataclysmic comet or asteroid explosion over top of Canada.

A comet/asteroid theory advanced by Arizona-based geophysicist Allen West in the past two years says that an object from space exploded just above the earth's surface at that time over modern-day Canada, sparking a massive shock wave and heat-generating event that set large parts of the northern hemisphere ablaze, setting the stage for the extinctions.

Now University of Cincinnati Assistant Professor of Anthropology Ken Tankersley, working in conjunction with Allen West and Indiana Geological Society Research Scientist Nelson R. Schaffer, has verified evidence from sites in Ohio and Indiana – including, locally, Hamilton and Clermont counties in Ohio and Brown County in Indiana – that offers the strongest support yet for the exploding comet/asteroid theory.

Samples of diamonds, gold and silver that have been found in the region have been conclusively sourced through X-ray diffractometry in the lab of UC Professor of Geology Warren Huff back to the diamond fields region of Canada.

The only plausible scenario available now for explaining their presence this far south is the kind of cataclysmic explosive event described by West's theory. "We believe this is the strongest evidence yet indicating a comet impact in that time period," says Tankersley.

Ironically, Tankersley had gone into the field with West believing he might be able to disprove West's theory.

Tankersley was familiar through years of work in this area with the diamonds, gold and silver deposits, which at one point could be found in such abundance in this region that the Hopewell Indians who lived here about 2,000 years ago engaged in trade in these items.

Prevailing thought said that these deposits, which are found at a soil depth consistent with the time frame of the comet/asteroid event, had been brought south from the Great Lakes region by glaciers.

"My smoking gun to disprove (West) was going to be the gold, silver and diamonds," Tankersley says. "But what I didn't know at that point was a conclusion he had reached that he had not yet made public – that the likely point of impact for the comet wasn't just anywhere over Canada, but located over Canada's diamond-bearing fields. Instead of becoming the basis for rejecting his hypothesis, these items became the very best evidence to support it."

Additional sourcing work is being done at the sites looking for iridium, micro-meteorites and nano-diamonds that bear the markers of the diamond-field region, which also should have been blasted by the impact into this region.

Much of the work is being done in Sheriden Cave in north-central Ohio's Wyandot County, a rich repository of material dating back to the Ice Age.

Tankersley first came into contact with West and Schaffer when they were invited guests for interdisciplinary colloquia presented by UC's Department of Geology this spring.

West presented on his theory that a large comet or asteroid, believed to be more than a mile in diameter, exploded just above the earth at a time when the last Ice Age appeared to be drawing to a close.

The timing attached to this theory of about 12,900 years ago is consistent with the known disappearances in North America of the wooly mammoth population and the first distinct human society to inhabit the continent, known as the Clovis civilization. At that time, climatic history suggests the Ice Age should have been drawing to a close, but a rapid change known as the Younger Dryas event, instead ushered in another 1,300 years of

glacial conditions. A cataclysmic explosion consistent with West's theory would have the potential to create the kind of atmospheric turmoil necessary to produce such conditions.

"The kind of evidence we are finding does suggest that climate change at the end of the last Ice Age was the result of a catastrophic event," Tankersley says.

Currently, Tankersley can be seen in a new documentary airing on the National Geographic channel. The film "Asteroids" is part of that network's "Naked Science" series.

The new discoveries made working with West and Schaffer will be incorporated into two more specials that Tankersley is currently involved with – one for the PBS series "Nova" and a second for the History Channel that will be filming Tankersley and his UC students in the field this summer. Another documentary, this one being produced by the Discovery Channel and the British public television network Channel 4, will also be following Tankersley and his students later this summer.

As more data continues to be compiled, Tankersley, West and Schaffer will be publishing about this newest twist in the search to explain the history of our planet and its climate.

Climate change is a favorite topic for Tankersley. "The ultimate importance of this kind of work is showing that we can't control everything," he says. "Our planet has been hit by asteroids many times throughout its history, and when that happens, it does produce climate change."

Is industrial pollution making America fat?

Studies link pervasive 'obesogens' to weight gain in frogs, mice

By Chris Lydgate

Exercise all you want, but it's pollution that may be a factor in weight gain, according to UC Irvine associate professor Bruce Blumberg.

Despite the nagging of diet experts, fitness instructors, public health officials, doctors, nurses and moms, the tide of obesity that has practically engulfed Western civilization over the past two decades shows no sign of reaching its ebb.

In the United States, the percentage of adults who are obese — defined by the National Institutes of Health as a body-mass index exceeding 30 — has doubled since 1990, climbing from 12 percent to a whopping 24 percent in 2005, closely tracking Oregon figures, according to the Oregon Health Division.

For the most part, the blame for the obesity epidemic has fallen on diet and exercise, with particular emphasis on familiar evils such as the proliferation of junk food, the advent of the remote control, trans fat, ever-longer commutes and even the disappearance of physical education in schools.

But now some researchers have identified a new suspect: pollution.

Attributing obesity to diet and exercise is "practically scientific dogma at this point," says Bruce Blumberg, associate professor of developmental and cell biology at UC Irvine. But, he continues, "diet and exercise are simply not adequate to explain the explosion of obesity in Western countries."

Instead, Blumberg believes the obesity epidemic actually is due, in part, to industrial pollution — specifically to low levels of toxic compounds he calls "obesogens."

Just as exposure to carcinogens can trigger cancer, Blumberg and other researchers say exposure to obesogens can trigger a dramatic increase in the amount of fat produced in a person's body, leading to excess weight and obesity.

The precise mechanism by which these obesogens operate remains dimly understood. They belong to a class of compounds known as "endocrine disrupters" because they block or pervert the operation of the hormones that govern crucial biological processes such as growth, reproduction, sexual development and behavior.

Five years ago, Blumberg was studying the biological effects of various marine pollutants — in particular, tributyl tin, or TBT, a pesticide notorious for its toxic properties, such as bizarre mutations in the shells of mollusks and the sex organs of sea snails.

Blumberg and his co-workers exposed female frogs to extremely low levels of TBT; as expected, TBT did indeed cause sexual mutation among frogs. But what was really striking, he says, was that the hapless amphibians got fat — really fat.

"To be honest, I will have to say we stumbled on this," he says.

Tiny doses had a big effect

Although most of the research on endocrine disrupters has focused on their potential effects on sexual development, fat production also is regulated by the hormone system and is, theoretically at least, just as susceptible to disruption.

Blumberg injected mice with TBT and observed similar results: fat rodents. Even more significant, the compound triggered obesity in ridiculously min-uscule quantities. In fact, Blumberg and his colleagues

demonstrated effects from TBT at 27 parts per billion — the rough equivalent of 4 tablespoons in an Olympic-sized swimming pool.

Blumberg concluded that the fattening effects of TBT and a group of similar compounds known as organotins are so profound that even trace amounts could trigger an increase in weight. “The introduction of organotins is likely to be a contributing factor to the obesity epidemic,” Blumberg says.

Toxicology experts concur that some compounds are so potent that they can indeed trigger changes at minute concentrations, at least in the test tube.

“It sounds absurd, but it’s not inconsistent with what we see in the lab,” says Fred Berman, director of the Toxicology Information Center at the Center for Research on Occupational and Environmental Toxicology at Oregon Health & Science University.

Organotins are everywhere

The disruptive effects of organotins stem from their propensity to stimulate a particular hormone receptor that plays a key role in maintaining the body’s metabolism, in effect telling the body which kind of cells are in short supply and need to be grown.

Organotins somehow encourage that receptor to manufacture fat cells — which in turn promotes that ominous abdominal bulge feared by statisticians and movie stars alike.

Organotins first came into widespread use in the 1960s in the shipbuilding industry, where they were mixed with paint to deter barnacles and mollusks from accumulating on the hulls of ships.

They also have been used as soil fungicides for crops such as nuts, potatoes, rice and celery; as “slimicide” to clean up the goop that accumulates in underground water wells; and in the manufacture of polyvinyl chloride, or PVC, a hard plastic found in drainpipes, vinyl flooring, window frames and hundreds of other places.

These widespread uses suggest several possible routes of human exposure, Blumberg says. Organotins may contaminate crops, seep into wells or leach into drinking water from PVC pipes.

Link to obesity stays unclear

It is worth pointing out, however, that little research has been conducted into actual levels of organotins in the average household.

Moreover, many basic questions about the link between organotins and fat remain unexplored — for example, whether people who might encounter high levels of organotins as a result of their occupation, such as farmworkers or shipyard workers, suffer higher rates of obesity.

The pernicious effects of organotins on the marine environment are well-established, and they now are banned as anti-barnacle agents.

Nonetheless, they continue to be used in agriculture and in the manufacture of plastics. “At my house, we minimize the use of plastic to store food,” Blumberg says. “We use glass or stainless steel instead, and in general we try to eat fresh, local, organic food with as little packaging as possible.”

OHSU’s Berman reckons that Blumberg’s research raises “a real concern” about the role of organotins and other possible pollutants in the obesity epidemic. “We need to be looking at this,” he says. “But we don’t know for sure. We need to do more studies to see if there is a real effect in the real world.”

Blumberg is careful to note that obesity is a complex phenomenon stemming from many factors, and that obesogens probably are only part of the story.

In addition, he points out that people who are exposed to obesogens are not doomed to a lifetime of corpulence — they simply have to work harder than others to shed weight.

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Ancient marine invertebrate diversity less explosive than thought

Diversity among the ancestors of such marine creatures as clams, sand dollars and lobsters showed only a modest rise beginning 144 million years ago with no clear trend afterwards, according to an international team of researchers. This contradicts previous work showing dramatic increases beginning 248 million years ago and may shed light on future diversity.

"Some of the time periods in the past are analogies for what is happening today from global warming," says Jocelyn Sessa, doctoral candidate in geosciences, Penn State. "Understanding what happened with diversity in the past can help us provide some prediction on how modern organisms will fare. If we know where we have been, we know something about where it will go."

Using contemporary statistical methods and the Paleobiology Database, the researchers report, in today's (July 4) issue of Science, a new diversity curve that shows that most of the early spread of invertebrates took place well before the Late Cretaceous, and that the net increase through the period since, is proportionately small relative to the 65 million years that elapsed.

One key to the new curve is the Paleobiology Database, (http://paleodb.org) housed at the National Center for Ecological Analysis and Synthesis, University of California, Santa Barbara. Previous research was based on databases of marine invertebrate fossils that recorded only the first occurrence of an organism and the last occurrence of the organism. There was no information in between for the organism.

"Over 30 years ago, researchers looked at the curve they had and considered that perhaps diversity did not increase at all," says Mark E. Patzkowsky, associate professor of geosciences. "What researchers saw was the diversity curve leveled off for quite some time and then took off exponentially. However, diversity results are strongly controlled by sampling techniques."

The new database allows researchers to standardize sample size because it includes multiple occurrences of each fossil. Researchers can randomly choose equal samples from equal time spans to create their diversity curve. This new curve uses 11 million-year segments, but the researchers hope to reduce the time intervals to 5 million years to match the interval of the previous curve, known as Sepkoski.

The data for this study contains 284,816 fossil occurrences of 18,702 genera that equals about 3.4 million specimens from 5384 literature sources. The old curve, developed by J. John Sepkoski Jr., used a database that contained only about 60,000 occurrences.

The researchers also looked at evenness in diversity. If there are 100 specimens divided into 10 time intervals, they could be divided with 10 individual specimens in each interval; or 91 specimens could be in one interval with one each in the remainder. The more even the distribution, the higher the evenness.

"Evenness says something about resource distribution," says Patzkowsky. "Much of invertebrate diversity has been attributed to diversity increase in the tropics, but the curve is not driven by that totally. It seems that 450 million years ago was not so different from today because it also contained more diversity in the tropics."

The major points of the Sepkoski curve are still seen in the new curve. Some things that are not seen, such as the decrease in diversity due to the Cretaceous Tertiary (KT) extinction 65 million years ago are not visible because of the scale of the intervals used. The extinction and recovery in the KT took less than 11 million years and so do not show. Some things not seen on the Sepkoski curve include a peak in the Permian. Also unexpected is that the diversity in the Jurassic (206 to 144 million years ago) is lower than diversity in the Triassic (248 to 206 million years ago), indicating a dip and rise in the diversity curve. The curve then rises in the Cretaceous and remains more or less flat after that. The previously thought exponential increase in diversity is not there.

"Comparing diversity through time is about how our world works, about the origin of species and how diversity changes with temperature," says Sessa. "If we think that the net increase over time will not get much greater, things are very different from if the diversity increases exponentially."

The National Science Foundation and NASA supported this research.

Beside Patzkowsky and Sessa, the Penn Staters on the project include Andrew Z. Krug, a recent Ph.D. recipient and postdoctoral fellow, University of Chicago, Thomas D. Olszewski, Ph.D. recipient and associate professor, Texas A&M, and former undergraduate students Philip S. Borkow, Hamilton Local School District, Columbus, Ohio, and Karen M. Layou, with work done at Penn State, University of Georgia and now visiting assistant professor, College of William and Mary.

Other researchers were John Alroy, lead author, University of California, Santa Barbara; Martin Aberhan, Wolfgang Kiessling, Benjamin Brenneis and Sabine Nurnberg, Humboldt University, Berlin; David J. Bottjer and Catherine M. Powers, University of Southern California; Nicole Bonusco, University of Southern California and University of California, Fullerton; Matthew E. Clapham, University of Southern California and University of California, Santa Cruz; Michael Foote, University of Chicago; Adam Tomasovych, University of Chicago and Slovak Academy of Sciences; Victoria L. Hanson, University of Chicago and University of Georgia; Carl Simpson, University of Chicago and Duke University; Shanan E. Peters, University of Chicago and University of Wisconsin-Madison; Franz T. Fursich, Friedrich-Alexander Universitat Erlangen, Germany; Peter J. Harries, University of South Florida; Austin J. W. Hendy, University of Cincinnati and Yale University; Arnold Miller and Chad A. Ferguson, University of Cincinnati; Steven M. Holland, University of Georgia; Linda C. Ivany, Syracuse University and Christy Visaggi, Syracuse University and University of North Carolina; Matthew A. Kosnik and Peter J. Wagner, Smithsonian Institution; Charles R. Marshall, Harvard University; Alistair J. McGowan, Natural History Museum, London, UK; Loic Villier, University of Provence, France; Leigh M. Fall, Texas A&M; Erin H. Leckey, University of Colorado.

Statins have unexpected effect on pool of powerful brain cells

Cholesterol-lowering drugs known as statins have a profound effect on an elite group of cells important to brain health as we age, scientists at the University of Rochester Medical Center have found. The new findings shed light on a long-debated potential role for statins in the area of dementia.

Neuroscientists found that statins, one of the most widely prescribed classes of medication ever used, have an unexpected effect on brain cells. Researchers looked at the effects of statins on glial progenitor cells, which help the brain stay healthy by serving as a crucial reservoir of cells that the brain can customize depending on its needs. The team found that the compounds spur the cells, which are very similar to stem cells, to shed their flexibility and become one particular type of cell.

The new findings come at a time of increasing awareness among neurologists and cardiologists of the possible effects of statins on the brain. Several studies have set out to show that statins provide some protection against dementia, but the evidence has been inconclusive at best. Meanwhile, there is some debate among physicians about whether statins might actually boost the risk of dementia. The new research published in the July issue of the journal Glia by Steven Goldman, M.D., Ph.D., and first author Fraser Sim, Ph.D., provides direct evidence for an effect of statins on brain cells.

"There has been a great deal of discussion about a link between statins and dementia, but evidence either way has been scant," said Goldman, a neurologist who led the team. "This new data provides a basis for further exploration.

"These findings were made through experiments done in cell culture using human brain cells and exposing them to doses of statins used widely in patients. But this research was not done in people. There are a great number of questions that need to be explored further before anyone considers changing the way statins are used," Goldman added.

Goldman's team is recognized as a leader identifying and directing the molecular signals that direct the development of stem cells and their daughter cells, known as progenitor cells. In this study, Sim ran a genomic screen to see which genes are more active in these cells compared to other brain cells. Sim and Goldman found several related to cholesterol, including the enzyme HMG-CoA reductase, which is central to making cholesterol and is the main target of statins.

"It was quite surprising that the cholesterol-signaling pathways are so active in these cells," Goldman said. "Since such signaling is blocked with compounds used literally by millions of patients every day, we decided to take a closer look."

The team measured the effects of two widely used statins, simvastatin and pravastatin, on glial progenitor cells, which can become either astrocytes or oligodendrocytes. The team looked at progenitor cells from 16 patients who had brain tissue removed during surgery to treat epilepsy, tumors, or vascular problems.

Scientists found that both compounds, when used at doses that mimic those that patients take, spur glial progenitor cells to develop into oligodendrocytes. For example, in one experiment, they found about five times as many oligodendrocytes in cultures of human progenitor cells exposed to pravastatin compared to cultures not exposed to the substance. Similarly, they found that the number of progenitor cells was just about one-sixth the level in cultures exposed to simvastatin compared to cultures not exposed to the compound.

To understand the process, think of a baseball team raising a group of great young prospects. They run fast, they throw hard, they hit well. Most teams will tailor their players to the positions the team needs – a few pitchers, for instance, and several batters. Any team that suddenly found itself with all pitchers or all hitters would be ill prepared to compete.

The Rochester team discovered that statins essentially push most of the raw talent in one direction.

Scientists don't really know the long-term effects of such a shift. Physicians are looking at statins as a possible treatment for multiple sclerosis, where the myelin coating that covers nerve cells in the central nervous system is damaged. Myelin is produced by oligodendrocytes – so spurring the development of oligodendrocytes might provide one way to reduce or repair the damage seen in M.S.

But the body maintains a pool of uncommitted glial progenitor cells for a reason. The body normally turns to that reservoir of cells when it needs to repair damage from a variety of causes, such as an infection, hemorrhage, a serious blow to the head, or inflammation within the brain, such as in patients with multiple sclerosis. No one knows the consequences if such cells weren't available when needed, though increased cognitive impairment might be one possibility.

"These are the cells ready to respond if you have a region of the brain that is damaged due to trauma, or lack of blood flow like a mini-stroke," said Sim, assistant professor of Neurology. "Researchers need to look very carefully at what happens if these cells have been depleted prematurely."

Glial progenitor cells are distributed throughout the brain and, according to Sim, make up about 3 percent of our brain cells. While true stem cells that can become any type of cell are very rare in the brain, their progeny, progenitor cells, are much more plentiful. They are slightly more specialized than stem cells but can still develop into different cell types.

The work may be relevant to drugs commonly used by diabetics as well. That's because the team discovered that a signaling molecule called PPAR gamma is central to the effect of statins on glial progenitor cells. When PPAR gamma was blocked, the statins no longer had the effect. Since PPAR gamma is the main target of diabetes medications such as Avandia and Actos, which trigger the molecule, Goldman said it's likely that those medications have the same effect on progenitor cells. He also noted that many patients are on both diabetes drugs and statins, which could increase the effect.

"Our results suggest the need for awareness of the possible toxicities accruing to long-term statin use, and identify one such potential toxicity, the premature differentiation and attendant long-term depletion of oligodendrocyte progenitor cells of the adult brain," conclude the authors in their Glia paper.

Besides Sim and Goldman, other authors include medical student Jennifer Lang, technical associate Tracy Ali, Cornell scientist Neeta Roy, and neurosurgeons Edward Vates, M.D., and Webster Pilcher, M.D. The National Institute of Neurological Disorders and Stroke and the National Multiple Sclerosis Society funded the work.

Geologists push back date basins formed, supporting frozen Earth theory

GAINESVILLE, Fla. --- Even in geology, it's not often a date gets revised by 500 million years.

But University of Florida geologists say they have found strong evidence that a half-dozen major basins in India were formed a billion or more years ago, making them at least 500 million years older than commonly thought. The findings appear to remove one of the major obstacles to the Snowball Earth theory that a frozen Earth was once entirely covered in snow and ice – and might even lend some weight to a controversial claim that complex life originated hundreds of million years earlier than most scientists currently believe.

"In modern geology, to revise the age of basins like this by 500 million years is pretty unique," says Joe Meert, a UF associate professor of geology.

Agreed Abhijit Basu, a professor of geological studies at Indiana University: "The required revision is enormous – 500 million years or about 11 percent of total Earth history."

Meert is one of eight authors of a paper on the research that recently appeared in the online edition of the journal Precambrian Research.

The Purana basins – which include the subject of the study, the Vindhyan basin – are located south of New Delhi in the northern and central regions of India. They are slight, mostly flat depressions in the Earth's crust that span thousands of square miles. For decades, Meert said, most geologists have believed the basins formed 500 million to 700 million years ago when the Earth's crust stretched, thinned and then subsided.

Meert said that date may have originated in early radiometric dating of sediment from the basin. Radiometric dating involves estimating age based on the decay or radioactive elements. Additionally, he said, apparent fossils retrieved from the basin seemed to have originated between 500 million and 700 million years ago.

The researchers were working on an unrelated project and had no intention of re-examining the basins' age. But then a UF graduate student, Laura Gregory, dated a kimberlite retrieved from the Vindhyan basin to about 1,073 million years ago. A kimberlite is a volcanic rock that contains diamonds.

Gregory also used paleomagnetism, a technique that estimates where rocks were formed by using the orientation of their magnetic minerals. Curious about whether the kimberlite results would apply more generally to the region, fellow UF graduate student Shawn Malone compared the kimberlite's orientation to other rocks from the Vindhyan basin. To his surprise, he found the orientations were virtually identical.

As a result, the geologists expanded the investigation, using a modified chain saw to drill wine-cork-sized cores out of dozens of rocks collected from 56 sites. Their contents all also had the same or very similar magnetic orientation, Meert said.

Much of the basins are composed of sediments that cannot be dated using any method. But Meert said the sediment also contains zircon, which can be dated using laser mass spectrometry – vaporizing tiny bits of the rocks with a laser, then analyzing their uranium and "daughter" lead contents to tease out their formation date based on rates of decay.

All the zircon the researchers tested originated 1,020 million years ago, Meert said.

The Snowball Earth theory posits that the Earth was covered in snow and ice from about 635 million to 700 million years ago. While much geological evidence has been found to support that theory worldwide, the Vindhyan and other Purana basins lacked numerous telltale signs, such as striated or scratched boulders formed when ice drags small pebbles over bedrock and boulder beds derived from glaciers known as tillites, Meert said. As a result, he said, the basins represented a prominent obstacle to the theory.

The new study removes that obstacle because it pushes back the origins of the basins to well before Snowball Earth would have occurred.

A 2007 study, conducted independently of the UF study and published in the Journal of Geology, dated rocks from another Purana basin to 1,020 million years ago, another 500-million-year revision. One of its authors was M.E. "Pat" Bickford, a professor emeritus at Syracuse University's department of earth sciences. Bickford said the revisions of the age of the Purana basins calls into question the hypothesis that they formed when the supercontinent Rodinia broke up. Rodinia is thought to have separated into the modern continents about 700 million years ago, but the revisions make the basins too old for that split, Bickford said.

The UF research could also support a Swedish paleontologist's controversial dating of multicellular creatures called Ediacarans from an older part of the basin to 1.6 billion years. But, said Meert, "Of all the implications of

this research, the notion that Ediacaran-like organisms may be much older than 580 million years is probably the most speculative."

Microwave ray gun controls crowds with noise

* 17:06 03 July 2008
* NewScientist.com news service
*** David Hambling**

A US company claims it is ready to build a microwave ray gun able to beam sounds directly into people's heads.

The device – dubbed MEDUSA (Mob Excess Deterrent Using Silent Audio) – exploits the microwave audio effect, in which short microwave pulses rapidly heat tissue, causing a shockwave inside the skull that can be detected by the ears. A series of pulses can be transmitted to produce recognisable sounds.

The device is aimed for military or crowd-control applications, but may have other uses.

Lev Sadovnik of the Sierra Nevada Corporation in the US is working on the system, having started work on a US navy research contract. The navy's report states that the effect was shown to be effective.

Scarecrow beam?

MEDUSA involves a microwave auditory effect "loud" enough to cause discomfort or even incapacitation. Sadovnik says that normal audio safety limits do not apply since the sound does not enter through the eardrums.

"The repel effect is a combination of loudness and the irritation factor," he says. "You can't block it out."

Sadovnik says the device will work thanks to a new reconfigurable antenna developed by colleague Vladimir Manasson. It steers the beam electronically, making it possible to flip from a broad to a narrow beam, or aim at multiple targets simultaneously.

Sadovnik says the technology could have non-military applications. Birds seem to be highly sensitive to microwave audio, he says, so it might be used to scare away unwanted flocks.

Sadovnik has also experimented with transmitting microwave audio to people with outer ear problems that impair their normal hearing.

Brain damage risk

James Lin of the Electrical and Computer Engineering Department at the University of Illinois in Chicago says that MEDUSA is feasible in principle.

He has carried out his own work on the technique, and was even approached by the music industry about using microwave audio to enhance sound systems, he told New Scientist.

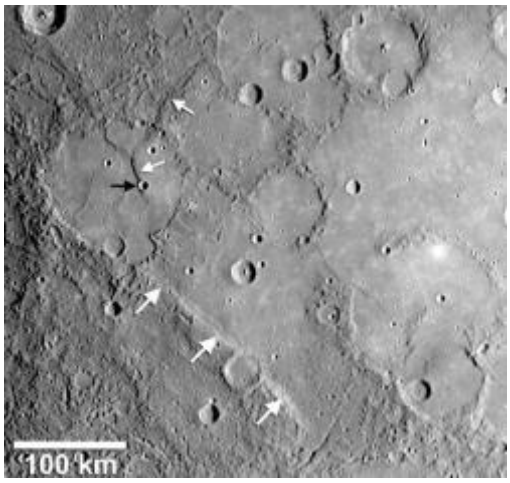
"But is it going to be possible at the power levels necessary?" he asks. Previous microwave audio tests involved very "quiet" sounds that were hard to hear, a high-power system would mean much more powerful – and potentially hazardous – shockwaves.

"I would worry about what other health effects it is having," says Lin. "You might see neural damage."

Sierra Nevada says that a demonstration version could be built in a year, with a transportable system following within 18 months. They are currently seeking funding for the work from the US Department of Defence.

Mercury: The incredible shrinking planet

* 20:44 03 July 2008
* NewScientist.com news service
*** Rachel Courtland**



The solar system's smallest planet has been shrinking at an unexpected rate, researchers announced on Thursday.

When NASA's Mariner 10 probe flew by Mercury in 1974 and 1975, it returned images of strange cliffs called 'scarps' that cut across all sorts of geological formations. That suggested that the planet's surface has contracted over time.

Now, pictures of Mercury's surface taken with NASA's Messenger spacecraft confirm that the crust appears to have buckled. In fact, the planet seems to have shrunk more than previously thought - and may still be shrinking.

The new result comes from an analysis of pictures snapped when the spacecraft whipped past the planet on 14 January and photographed a previously unseen 20% of the surface.

Scarps that cut across the surface of Mercury suggest the planet has been shrinking **(Image: Science/AAAS)**

In order for such shrinking to take place, Mercury must have had a molten, liquid core that has cooled and contracted over time.

That may settle a debate that has raged for more than 30 years about the planet's magnetic field. Researchers have wondered whether the field is more like Earth's, fed by a dynamo of circulating fluid, or like that of Mars, which hosts solid, magnetised rocks.

Liquid motion

In 2007, a team bounced radar signals off Mercury, revealing slight variations in the speed of the planet's rotation that suggested the planet's core is partly liquid.

Now Messenger researchers say additional images of scarps, along with a better map of the planet's magnetic field, show Mercury has a constitution similar to Earth's.

"Both of these results point therefore to an active source for Mercury's magnetic field, a dynamo stirred by motions in Mercury's fluid outer core," says the mission's chief scientist, Sean Solomon of the Carnegie Institution of Washington in Washington DC, US.

Mariner 10 images suggested the planet's radius, which is roughly 2400 kilometres, has shrunk by 1 to 2 km since its formation more than 4 billion years ago. "What Messenger is showing us is that's an underestimate," says Solomon.

The new scarp images, which were taken from a better angle relative to the Sun than Mariner 10, show the planet seems to be shrinking by at least a third more than previously thought.

A second Messenger flyby on 6 October, which will image another 30% of the planet's surface, could show shrinkage that is even more dramatic, says Solomon.

Further study may help date the scarps, revealing whether Mercury is still shrinking or has stopped.

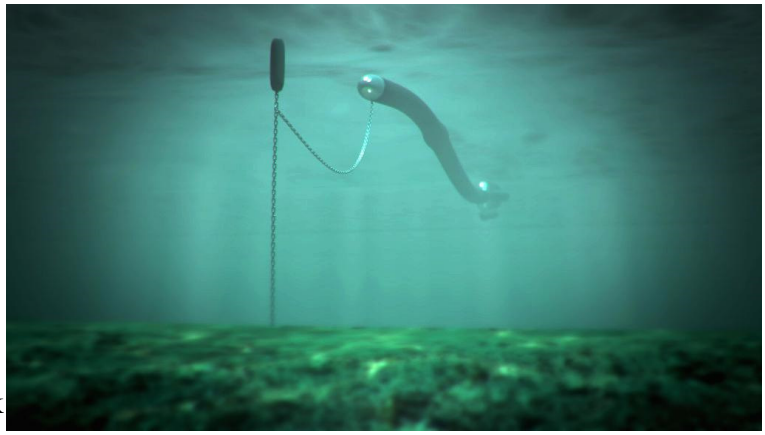
Journal reference: Science (vol 321)

Giant rubber snake could be the future of wave power

* 14:09 04 July 2008

* NewScientist.com news service

*** Tom Simonite**



A rubbery cylinder filled with seawater is said to efficiently harvest the energy from ocean waves

A giant rubber snake could be the future of renewable energy. The rippling "Anaconda" produces electricity as it is squeezed by passing waves. Its developers say it would produce more energy than existing wave-energy devices and be cheaper to maintain.

Retired physicist Francis Farley and Rod Rainey of Atkins Global dreamed up a flexible tube filled with seawater and sealed at both ends like a giant sausage. The structure streams out in the waves like a windsock pushed by the wind.

The passage of each wave squeezes the rubber and produces a bulging pressure wave that travels down its length. When the bulge reaches the end it sets turbines spinning to generate electricity.

Slippery customer

Eventually, full-scale versions should be 7 metres across, 200 m long and be anchored at one end in water between 40 m and 100 m deep.

For now, however, engineers John Chaplin and Grant Hearn at the University of Southampton are testing mini Anacondas, a few metres long, in a wave tank. "The top barely breaks the surface, and you can see the bulges moving down the tube," says Chaplin.

"In engineering terms, it is unlike any other offshore structure," he told New Scientist. "It's not a solid structure like an oil platform and it doesn't behave like a boat either."

Preliminary results are promising, says Chaplin. By tuning the diameter, flexibility and thickness of the rubber tube it is possible to make the Anaconda's pressure bulges travel at roughly the same speed as the waves outside. As a result they gradually gather more energy from the waves as they travel down the tube.

Snake scales

A full-scale device should produce 1 megawatt – enough to power around 200 houses.

By comparison, each jointed steel cylinder of the Pelamis wave power system which is being trialled in Portugal generates just 0.75 MW.

Anaconda's unique design should also handle the greatest challenge of wave energy better. "The ocean is a very hostile environment," says Chaplin. "The structure has got to be there and still working after the largest storms." What's more, saltwater corrodes metal structures, making maintenance costs high, he says.

A rubber structure with few mechanical parts exposed to the sea should be more resilient. Chaplin hopes to have a one-third scale model for testing in the sea next year and says full-scale Anacondas could be commercially available in five years.

Cleaner fish calms predators with caresses

* 16:26 04 July 2008
* NewScientist.com news service
*** Nora Schultz**

(Image: Alexandra S. Grutter)

Peacemakers should look to the cleaner fish as a role model. In the coral reef world at least, all it takes to keep an aggressive predator in check and bystanders safe is good service and a gentle rub.

Cleaner fish remove and eat the parasites off other fish, exchanging a grooming service for a tasty meal.

Researchers have previously shown that cleaners who enhance their service by touching the fish they are cleaning with their fins benefit from more cooperative clients. This is especially helpful if the customer is a predator that could attack the cleaner.

But now it seems that the calming effect of the cleaner fish's touch has wider repercussions. It makes hunters so mellow that it transforms the cleaning station into a safe haven for other fish, says Redouan Bshary at the University of Neuchatel in Switzerland.

Safe haven

"I've always marvelled at prey fish seeming so relaxed and non-vigilant at cleaning stations, even when lots of predators are around. A cleaning station should be a marvellous place for hunters to strike, but it never happens," Bshary says.

To test if cleaner fish influenced predators' behaviour towards prey, Bshary and colleagues from the University of Queensland in Brisbane, Australia, built mini coral-reefs in aquaria and observed the interactions between cleaners, their predatory and non-predatory clients and potential prey species that were not clients of the cleaners.

They found that predators in aquaria with cleaners chased prey two thirds less often than those in aquaria without a cleaner fish.

This peacefulness was not just a result of them having had less time to hunt because they were busy being groomed – their aggression was suppressed even when they were not being tended to by the cleaner fish.

Swim strokes

The number of chases also decreased the longer a cleaner spent touching a predator with its fins, and predators received at least three times as many touches as any of the other clients.

Jens Krause at the University of Leeds says that interacting with a cleaner fish may trigger a simple behavioural switch in the predators, causing them to stop foraging.

"But it is still unclear if the cleaners actively mediate because they benefit from an overall peaceful environment, or if other fish receive protection simply as a side effect of the cleaner fish protecting itself," he says.

Bshary and colleagues want to test this by looking at whether cleaner fish stroke predators more when potential prey species are nearby. They also intend to measure the predators' heart rate with hydrophones to see if the cleaners' massage has a physiological effect.

Journal reference: Behavioral Ecology (DOI: 10.1093/beheco/arn067)