

Yo-yo dieting appears to be healthier than lifelong obesity

A new study comparing lifelong obesity with the weight fluctuations of "yo-yo dieting" suggests it is better to attempt to lose weight despite repeated failures at keeping the weight off than to not diet and remain obese.

"It is clear that remaining on a stable, healthy diet provides the best outcome for health and longevity," said the study's principal investigator, Edward List, PhD, a scientist at Ohio University, Athens. "However, obese individuals commonly weight cycle—they have repeated intentional weight loss followed by weight regain, often called yo-yo dieting. While yo-yo dieting is thought to be harmful, there is little hard scientific evidence to support that." The results will be presented Monday at The Endocrine Society's 93rd Annual Meeting in Boston.

To determine the long-term health effects of yo-yo dieting, List and his collaborators performed what they call "the first controlled study of a yo-yo diet regimen used for an entire life span." Because of the challenges of performing a long-term controlled feeding study in humans, they used mice to test whether weight fluctuation due to yo-yo dieting is as unhealthy as lifelong obesity.

Thirty mice, in groups of 10 each, received one of three diets: high fat, low fat or a yo-yo diet, consisting of four weeks of the high-fat diet followed by four weeks of the low-fat diet. The mice stayed on their respective diets throughout their life span. Measures of health, including body weight, body fat and blood glucose (sugar) levels, were obtained.

List said the yo-yo diet resulted in large fluctuations in these health measures, decreasing during the low-fat diet and increasing to a diabetic state during the high-fat diet. When health measures during the high-fat and low-fat diet regimens of the yo-yo diet group were averaged, their "average health" was improved compared with obese mice that stayed on the high-fat diet, he reported. Compared with the mice fed the high-fat diet, mice on the yo-yo diet lived nearly 35 percent longer.

"Surprisingly, the mice on the yo-yo diet had a similar life span to that of the low-fat-fed group," List said.

These findings are important in light of the growing epidemic of obesity around the world, he stated.

"The fear of negative health consequences due to weight cycling may be overemphasized," List concluded. "From our study, it appears that it is better to continue to encourage weight loss regardless of the number of attempts and failures."

Funding for this study came from the Ohio University Diabetes Research Initiative and the Provost's Undergraduate Research Fund, AMVETS and the National Institutes of Health.

Deciding to stay or go is a deep-seated brain function

DURHAM, N.C. – *Birds do it. Bees do it. Even little kids picking strawberries do it.*

Every creature that forages for food decides at some point that the food source they're working on is no richer than the rest of the patch and that it's time to move on and find something better.

This kind of foraging decision is a fundamental problem that goes far back in evolutionary history and is dealt with by creatures that don't even have proper brains, said Michael Platt, a professor of neurobiology and director of the Center for Cognitive Neuroscience at Duke University.

Platt and his colleagues now say they've identified a function in the primate brain that appears to be handling this stay-or-go problem. They have found that the dorsal anterior cingulate cortex (ACC), an area of the brain known to operate while weighing conflicts, steadily increases its activity during foraging decisions until a threshold level of activity is reached, whereupon the individual decides it's time to move on.

In lab experiments with rhesus macaque monkeys, Platt and postdoctoral fellows Benjamin Hayden and John Pearson put the animals through a series of trials in which they repeatedly had to decide whether to stay with a source that was giving ever-smaller squirts of fruit juice, or move to another, possibly better, source. The animals were merely gazing at a preferred target on a display screen, not moving from one tree to the next, but the decision-making process should be the same, Platt said.

For the other variable in this basic equation, travel time, the researchers added delays when monkeys chose to leave one resource and move to another, simulating short and long travel times.

As the monkeys repeatedly chose to stay with their current source or move to another, the researchers watched a small set of neurons within the anterior cingulate cortex fire with increasing activity for each decision. The rate of firing in this group of neurons grew until a threshold was reached, at which time the monkey immediately decided to move on, Platt said. "It is as if there is a threshold for deciding it's time to leave set in the brain," he said.

When the researchers raised the "travel time" to the next foraging spot in the experiment, it raised the decision-making threshold, Platt said.

This all fits with a 1976 theory by evolutionary ecologist Eric Charnov, called the Marginal Value Theorem, Platt said. It says that all foragers make calculations of reward and cost that tell them to leave a patch when their intake diminishes to the average intake rate for the overall environment. That is, one doesn't pick a blueberry bush until it's bare, only until it looks about as abundant as the bushes on either side of it. Shorter travel time to the next patch means it costs less to move, and foragers should move more easily. This theorem has been found to hold in organisms as diverse as worms, bees, wasps, spiders, fish, birds, seals and even plants, Platt said. "This is a really fundamental solution to a fundamental problem," Platt said.

Platt said the work also relates to recent papers on the Web-browsing habits of humans. In the case of Internet users, the cost of travel time translates to download speed. The faster the downloads, the quicker browsers are willing to forage elsewhere, Platt said.

They aren't sure yet where the brain's signaling goes after the stay-or-go threshold in the ACC is reached. Platt believes this kind of "integrate-to-threshold" mechanism would be a good way to handle a lot of functions in the brain and may be found in other kinds of systems. This particular threshold in the ACC might also be a way to explain maladaptive behaviors like attention deficit, in which a person decides to move on constantly, or compulsive behavior, in which a person can't seem to move on at all, he said.

The research appears online in Nature Neuroscience, June 5, 2011. It was supported by the National Institutes of Health and a fellowship from the Tourette Syndrome Association.

CITATIONS: "Neuronal basis of sequential foraging decisions in a patchy environment," Benjamin Y. Hayden, John M.

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http://www.eurekalert.org/pub_releases/2011-06/uoc-nds060611.php

New data still have scientists in dark over dark matter

A dark-matter experiment deep in the Soudan mine of Minnesota now has detected a seasonal signal variation similar to one an Italian experiment has been reporting for more than a decade.

The new seasonal variation, recorded by the Coherent Germanium Neutrino Technology (CoGeNT) experiment, is exactly what theoreticians had predicted if dark matter turned out to be what physicists call Weakly Interacting Massive Particles (WIMPs). "We cannot call this a WIMP signal. It's just what you might expect from it," said Juan Collar, associate professor in physics at the University of Chicago. Collar and John Orrell of Pacific Northwest National Laboratory, who lead the CoGeNT collaboration, are submitting their results in two papers to Physical Review Letters.

WIMPs might have caused the signal variation, but it also might be a random fluctuation, a false reading sparked by the experimental apparatus itself or even some exotic new phenomenon in atomic physics, Collar said. Dark matter accounts for nearly 90 percent of all matter in the universe, yet its identity remains one of the biggest mysteries of modern science. Although dark matter is invisible to telescopes, astronomers know it is there from the gravitational influence it exerts over galaxies.

Theorists had predicted that dark matter experiments would detect an annual modulation because of the relative motion of the Earth and sun with respect to the plane of the Milky Way galaxy.

The sun moves in the plane of the galaxy on the outskirts of one of its spiral arms at a speed of 220 kilometers per second (136 miles per second). The Earth orbits the sun at 30 kilometers per second (18.5 miles per second). During winter, Earth moves in roughly the opposite direction of the sun's movement through the galaxy, but during summer, their motion becomes nearly aligned in the same direction. This alignment increases Earth's net velocity through a galactic halo of dark matter particles, whose existence scientists have inferred from numerous astronomical observations.

Like a cloud of gnats

WIMPs would be moving in random directions in this halo, at velocities similar to the sun's. "You find yourself in a situation similar to a car moving through a cloud of gnats," Collar explained. "The faster the car goes, the more gnats will hit the front windshield."

CoGeNT seems to have detected an average of one WIMP particle interaction per day throughout its 15 months of operation, with a seasonal variation of approximately 16 percent. Energy measurements are consistent with a WIMP mass of approximately 6 to 10 times the mass of a proton.

These results could be consistent with those of the Italian DARK MATTER (DAMA) experiment, which has detected a seasonal modulation for years. "We are in the very unfortunate situation where you cannot tell if we are barely excluding DAMA or barely in agreement. We have to clarify that," Collar said.

In particle physics, he further cautioned, agreement between two or three experiments doesn't necessarily mean much. The pentaquark is a case in point. Early this century, approximately 10 experiments found hints of evidence for the pentaquark, a particle consisting of five quarks, when no other known particle had more than three. But as time went on, new experiments were unable to see it. "It's just incredible," said UChicago physics Professor Jonathan Rosner. "People still speculate on whether it's real."

Collar and his colleagues have calculated the probability that their finding is a fluke to be five-tenths of a percent, or 2.8 sigma in particle physics parlance. "It's not an exact science yet, unfortunately," Collar said. "But with the information we have, the usual set of assumptions that we make about the halo and these particles, their behavior in this halo, things seem to be what you would expect."

Other dark-matter experiments, including Xenon100, have not detected the seasonal signal that CoGeNT and DAMA have reported. "If you really wanted to see an effect, you could argue that the Xenon100 people don't have the sensitivity to Juan's result," said Rosner, who is not a member of the CoGeNT collaboration. "On the other hand, they've done a number of studies of what their sensitivity is at low energies and they believe they're excluding this result."

Interrupted by fire

CoGeNT operated from December 2009 until interrupted by a fire in the Soudan mine in March 2011. Fifteen months of data collection is a relatively brief period for a dark-matter experiment. In fact, Collar and his colleagues decided to examine the data now only because the fire had stopped the experiment, at least temporarily. The fire did not directly affect the experiment, but the CoGeNT team has not been able to examine the detector because of clean-up efforts. The detector may no longer work, or if it does work, it may now have different properties.

"This effect that we're seeing is touch-and-go. It's something where you have to keep the detector exquisitely stable," Collar said. If a single key characteristic of the detector has changed, such as its electronic noise, "We may be unable to look for this modulation with it from now on."

The putative mass of the WIMP particles that CoGeNT possibly has detected ranges from six to 10 billion electron volts, or approximately seven times the mass of a proton. "To look for WIMPs 10 times heavier is hard enough. If they're this light, it becomes a nightmare," Collar said.

Related links [Q&A with Juan Collar on the hunt for dark matter \(June 6, 2011\) http://www.kavlifoundation.org/science-spotlights/chicago-dark-matter-modulation-collar](http://www.kavlifoundation.org/science-spotlights/chicago-dark-matter-modulation-collar)

Dark matter search plunges physicists to new depths (Aug. 11, 2010) http://news.uchicago.edu/article/2010/08/10/dark-matter-search-plunges-physicists-new-depths

http://www.eurekalert.org/pub_releases/2011-06/afps-bin060611.php

Be it numbers or words -- the structure of our language remains the same

It is one of the wonders of language: We cannot possibly anticipate or memorize every potential word, phrase, or sentence.

Yet we have no trouble constructing and understanding myriads of novel utterances every day. How do we do it? Linguists say we naturally and unconsciously employ abstract rules—syntax.

How abstract is language? What is the nature of these abstract representations? And do the same rules travel among realms of cognition? A new study exploring these questions—by psychologists Christoph Scheepers, Catherine J. Martin, Andriy Myachykov, Kay Teevan, and Izabela Viskupova of the University of Glasgow, and Patrick Sturt of the University of Edinburgh—makes what Scheepers calls "a striking new finding": The process of storing and reusing syntax "works across cognitive domains."

More specifically: "The structure of a math equation correctly solved is preserved in memory and determines the structuring of a subsequent sentence that a person has to complete." Neuroscientists have found evidence suggesting a link between math and language, "but this is the first time we've shown it in a behavioral setup."

The findings will be published in an upcoming issue of *Psychological Science*, a journal of the Association for Psychological Science.

The study made use of a cognitive process called structural priming. Simply put, if you use a certain kind of structure in one sentence, you're likely to use it again in a subsequent sentence. To find out how abstract—and cognitively general—this process is, the experimenters gave native English-speaking students a pencil-and-paper test containing a series of math problems paired with incomplete sentences.

Each math problem was structured in one of three ways. With "high-attachment" syntax, the final operation of the problem applied to a large "chunk" of the earlier part. For instance: $80 - (5 + 15) / 5$, where the final division ($/ 5$) applies to the previous addition term ($5 + 15$). With "low-attachment" syntax—say, $80 - 5 + 15 / 5$ —the final operation applied to a smaller previous chunk. A third category—"baseline" problems like $80 - 5$ —implied neither high nor low attachment.

After each equation, the participant was given a sentence fragment that could be completed with either high or low attachment syntax. For instance – The tourist guide mentioned the bells of the church that ... A high-attachment ending would refer to the entire phrase the bells of the church and might finish with "that chime hourly." Low attachment would link only the church to the completed final clause—say, "that stands on a hill."

The subjects were variously successful in solving the problems. Their choice of high or low attachment sentence completions also revealed complexities—some perhaps related to the preference in English for low-attachment syntax. Still, in significant numbers, high-attachment math problems primed high-attachment sentence completions, and low-attachment problems made low-attachment completions likely.

What does all this mean? Our cognitive processes operate "at a very high level of abstraction," the authors write. And those abstractions may apply in similar fashion to all kinds of thinking—in numbers, words, or perhaps even music.

The APS journal Psychological Science is the highest ranked empirical journal in psychology. For a copy of the article "Structural Priming across Cognitive Domains: From Simple Arithmetic to Relative Clause Attachment" and access to other Psychological Science research findings, please contact Divya Menon at 202-293-9300 or dmenon@psychologicalscience.org. http://www.eurekalert.org/pub_releases/2011-06/su-sc060611.php

Stanford climate scientists forecast permanently hotter summers

The tropics and much of the Northern Hemisphere are likely to experience an irreversible rise in summer temperatures within the next 20 to 60 years if atmospheric greenhouse gas concentrations continue to increase, according to a new climate study by Stanford University scientists.

In the study, the Stanford team concluded that many tropical regions in Africa, Asia and South America could see "the permanent emergence of unprecedented summer heat" in the next two decades. Middle latitudes of Europe, China and North America – including the United States – are likely to undergo extreme summer temperature shifts within 60 years, the researchers found. The results will be published later this month in the journal *Climatic Change*.

"According to our projections, large areas of the globe are likely to warm up so quickly that, by the middle of this century, even the coolest summers will be hotter than the hottest summers of the past 50 years," said the study's lead author, Noah Diffenbaugh, an assistant professor of environmental Earth system science and fellow at the Woods Institute for the Environment at Stanford. The study is co-authored by Stanford research assistant Martin Scherer.

"When scientists talk about global warming causing more heat waves, people often ask if that means that the hottest temperatures will become 'the new normal,'" Diffenbaugh said. "That got us thinking – at what point can we expect the coolest seasonal temperatures to always be hotter than the historically highest temperatures for that season?"

Climate models, past and future

To determine the seasonal impact of global warming in coming decades, Diffenbaugh and Scherer analyzed more than 50 climate model experiments –including computer simulations of the 21st century when global greenhouse gas concentrations are expected to increase, and simulations of the 20th century that accurately "predicted" the Earth's climate during the last 50 years. The analysis revealed that many parts of the planet could experience a permanent spike in seasonal temperatures within 60 years.

"We also analyzed historical data from weather stations around the world to see if the projected emergence of unprecedented heat had already begun," Diffenbaugh said. "It turns out that when we look back in time using temperature records, we find that this extreme heat emergence is occurring now, and that climate models represent the historical patterns remarkably well."

According to both the climate model analysis and the historical weather data, the tropics are heating up the fastest. "We find that the most immediate increase in extreme seasonal heat occurs in the tropics, with up to 70 percent of seasons in the early 21st century (2010-2039) exceeding the late-20th century maximum," the authors wrote.

Tropical regions may see the most dramatic changes first, but wide swaths of North America, China and Mediterranean Europe are also likely to enter into a new heat regime by 2070, according to the study.

Environmental impact

This dramatic shift in seasonal temperatures could have severe consequences for human health, agricultural production and ecosystem productivity, Diffenbaugh said. As an example, he pointed to record heat waves in Europe in 2003 that killed 40,000 people. He also cited studies showing that projected increases in summer temperatures in the Midwestern United States could reduce the harvest of staples, such as corn and soybeans, by more than 30 percent.

Diffenbaugh was surprised to see how quickly the new, potentially destructive heat regimes are likely to emerge, given that the study was based on a relatively moderate forecast of greenhouse gas emissions in the 21st century.

"The fact that we're already seeing these changes in historical weather observations, and that they match climate model simulations so closely, increases our confidence that our projections of permanent escalations in seasonal temperatures within the next few decades are well founded," Diffenbaugh said.

The research was supported by the National Science Foundation, the Department of Energy, the National Institutes of Health and the World Bank.

This article was written by Donna Hesterman, a science-writer intern at the Woods Institute for the Environment at Stanford University.

<http://www.newscientist.com/article/dn20546-early-americans-helped-colonise-easter-island.html>

Early Americans helped colonise Easter Island

*** 22:34 06 June 2011 by Michael Marshall**

South Americans helped colonise Easter Island centuries before Europeans reached it.

Clear genetic evidence has, for the first time, given support to elements of this controversial theory showing that while the remote island was mostly colonised from the west, there was also some influx of people from the Americas.

Easter Island is the easternmost island of Polynesia, the scattering of islands that stretches across the Pacific. It is also one of the most remote inhabited islands in the world.

So how did it come to be inhabited in the first place? Genetics, archaeology and linguistics all show that as a whole, Polynesia was colonised from Asia, probably from around Taiwan. The various lines of evidence suggest people began migrating east around 5500 years ago, reached Polynesia 2500 years later, before finally gaining Easter Island after another 1500 years.

But the Norwegian adventurer Thor Heyerdahl thought otherwise. In the mid-20th century, he claimed that the famous Easter Island statues were similar to those at Tiahuanaco at Lake Titicaca in Bolivia, so people from South America must have travelled west across the Pacific to Polynesia. His famous Kon-Tiki expedition, in which he sailed a balsa wood raft from Peru to the Tuamotu islands of French Polynesia, showed that the trip could have been made. But if it was made, no trace remained.

Now Erik Thorsby of the University of Oslo in Norway has found clear evidence to support elements of Heyerdahl's hypothesis. In 1971 and 2008 he collected blood samples from Easter Islanders whose ancestors had not interbred with Europeans and other visitors to the island.

Thorsby looked at the HLA genes, which vary greatly from person to person. Most of the islanders' HLA genes were Polynesian, but a few of them also carried HLA genes only previously found in Native American populations.

Genetic shuffling

Because most of Thorsby's volunteers came from one extended family, he was able to work out when the HLA genes entered their lineage. The most probable first known carrier was a woman named Maria Aquala, born in 1846. Crucially, that was before the slave traders arrived in the 1860s and began interbreeding with the islanders.

But the genes may have been around for longer than that. Thorsby found that in some cases the Polynesian and American HLA genes were shuffled together, the result of a process known "recombination". This is rare in HLA genes, meaning the American genes would need to be around for a certain amount of time for it to happen. Thorsby can't put a precise date on it, but says it is likely that Americans reached Easter Island before it was "discovered" by Europeans in 1722.

Thorsby says there may have been a Kon-Tiki-style voyage from South America to Polynesia. Alternatively, Polynesians may have travelled east to South America, and then returned. There is already evidence for that: chicken bones found in Chile turned out to be Polynesian, so we know that the eastward journey did happen at some stage.

However, Thorsby's findings don't mean that Heyerdahl's ideas have been vindicated. The first settlers to Polynesia came from Asia, and they made the biggest contribution to the population. "Heyerdahl was wrong," Thorsby says, "but not completely."

The work was presented at a Royal Society discussion meeting on human evolution in London today.

Journal references: Tissue Antigens, DOI: 10.1111/j.1399-0039.2006.00717.x and 10.1111/j.1399-0039.2009.01233.x

Human Ancestors in Eurasia Earlier than Thought

Stone fragments found in Georgia suggest might have evolved outside Africa.

By Matt Kaplan of Nature magazine

Archaeologists have long thought that *Homo erectus*, humanity's first ancestor to spread around the world, evolved in Africa before dispersing throughout Europe and Asia. But evidence of tool-making at the border of Europe and Asia is challenging that assumption.

Reid Ferring, an anthropologist at the University of North Texas in Denton, and his colleagues excavated the Dmanisi site in the Caucasus Mountains of Georgia. They found stone artifacts--mostly flakes that were dropped as hominins knapped rocks to create tools for butchering animals--lying in sediments almost 1.85 million years old. Until now, anthropologists have thought that *H. erectus* evolved between 1.78 million and 1.65 million years ago--after the Dmanisi tools would have been made.

Furthermore, the distribution of the 122 artifacts paints a picture of long-term occupation of the area. Instead of all the finds being concentrated in one layer of sediment, which would indicate that hominins visited the site briefly on one occasion, the artifacts are spread through several layers of sediment that span the period between 1.85 million and 1.77 million years ago. The findings are published June 6 in Proceedings of the National Academy of Sciences.

"This is indeed suggestive of a sustained regional population which had successfully adapted to the temperate environments of the southern Caucasus," explains Wil Roebroeks, an archaeologist at Leiden University in the Netherlands.

Eurasian ancestry?

The presence of a tool-using population on the edge of Europe so early hints that the northern continent, rather than Africa, may have been the evolutionary birthplace of *H. erectus*. Unfortunately, the fossils of the hominins responsible for making the tools are not proving very helpful to the debate.

Fossilized bone fragments found in the same sedimentary layers as the Dmanisi artifacts are too weathered to be identified as belonging to any one species, so it is impossible to say for sure whether the tools were made by *H. erectus*.

Neither do fossil skulls previously retrieved from later sediments at the site help to resolve the controversy. These fossils, dating from 1.77 million years ago, had brains between 600 and 775 cubic centimeters in volume, whereas *H. erectus* is generally thought to have had an average brain size of around 900 cubic centimeters. For comparison, modern humans have a brain capacity of around 1,350 cubic centimeters. "Many people call those Dmanisi fossils the earliest *H. erectus*, but there is still frequent debate about this," explains Ferring.

There and back again

Even if the ancient inhabitants of the Dmanisi site were not early members of *H. erectus*, there is still a problem: anthropologists have previously thought that no hominins existed outside of Africa as early as 1.85 million years ago.

"Anthropology textbooks of the 1990s often showed maps with large arrows indicating migration of early *H. erectus* from its inferred core area of eastern Africa to other parts of the Old World," explains Roebroeks. The findings in Dmanisi make such an explanation look faulty.

Ferring and his colleagues propose that some ancestors of *H. erectus* might have traveled to Asia and possibly Europe, done a bit of evolving, then wandered back to Africa.

"Remember, it would not have been obvious to the hominins they were leaving Africa. There were no signs saying, 'You are leaving Africa now--come and visit us again!'" says Bernard Wood, an anthropologist at the George Washington University in Washington, D.C. But Wood admits that it is unclear why the hominins might have made these movements. "It perplexes me," he says.

Ferring suggests that ancient hominins might have been following their food source--animals. "My hunch is that the migrations relate to the rise of carnivory and a sudden flexibility to live and eat meat anywhere," he says. Vegetarians, he explains, are limited to the specific plants that sustain them and cannot travel from tropics to deserts to mountains nearly as easily as predators can. Wood agrees. "My guess is that hominins were following game," he says.

Other possibilities also exist. "We tend to think of hominins as living in a disease-free world, but maybe they were eliminated in some places by an epidemic, and the only healthy ones left were at the edges of their distribution," who could then move back into the vacated areas, says Wood.

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http://www.eurekalert.org/pub_releases/2011-06/uos-ep1060711.php

Experts prove link between phosphate intake and heart disease

Lowering phosphate intake in humans can reduce heart disease, according to research by experts at the University of Sheffield.

This is the first time the connection between a high phosphate diet and atherosclerosis - the cause of heart disease - has been proven. The findings have been published in *Arteriosclerosis, Thrombosis and Vascular Biology* (2 June 2011).

The research, which was funded by the Sheffield Kidney Association and the National Institute for Health Research, has shown that cholesterol deposits in the wall of arteries are increased following a higher phosphate diet. This leads to narrowing of the arteries, which is the cause of most heart attacks and strokes.

As a result, the research demonstrates the importance of reducing phosphate levels in the human diet or possibly using drugs called binders or other agents that stop phosphate being absorbed. Food high in phosphate includes biscuits, cakes, sweets, dairy products and meats such as offal and veal.

Dr Tim Chico from the University's Department of Cardiovascular Science, who led the research, said: "This is a very early, but exciting finding, as it suggests that by reducing the amount of phosphate in the blood we may have discovered a new approach to reducing heart disease. We're now hoping to extend our research further and look into developing new treatments to help reduce phosphate levels in the bloodstream."

Notes to editors: To read the full paper by Timothy Ellam, Martin Wilkie, Janet Chamberlain, David Crossman, Richard Eastell, Sheila Francis, and Timothy J.A. Chico, visit: <http://atvb.ahajournals.org/cgi/reprint/ATVBAHA.111.231001v1>

http://www.eurekalert.org/pub_releases/2011-06/aga-cdi060711.php

Coffee drinking improves hepatitis C treatment response

Advanced hepatitis C patients with chronic liver disease may benefit from drinking coffee during treatment, according to a new study in *Gastroenterology*, the official journal of the American Gastroenterological Association (AGA) Institute.

Patients who received peginterferon plus ribavirin treatment and who drank three or more cups of coffee per day were two times more likely to respond to treatment than non-drinkers.

"Coffee intake has been associated with a lower level of liver enzymes, reduced progression of chronic liver disease and reduced incidence of liver cancer," said Neal Freedman, PhD, MPH, of the National Cancer Institute and lead author of this study. "Although we observed an independent association between coffee intake and virologic response to treatment, this association needs replication in other studies."

Among non-drinkers, 46 percent had an early virologic response; 26 percent had no detectable serum hepatitis C virus (HCV) ribonucleic acid at week 20; 22 percent had no detectable serum at week 48; and 11 percent had a sustained virologic response. In contrast, the corresponding proportions for those who drank three or more cups of coffee per day were 73 percent, 52 percent, 49 percent and 26 percent, respectively.

Approximately 70 to 80 percent of individuals exposed to HCV become chronically infected. Worldwide, these individuals are estimated to number between 130 and 170 million. Higher coffee consumption has been associated with slower progression of pre-existing liver disease and lower risk of liver cancer. However, the relationship with response to anti-HCV treatment had not been previously evaluated. Treatment with peginterferon and ribavirin resolves chronic hepatitis C in about half of patients. It is unknown whether coffee will improve response with the addition of new drugs that were recently approved for use in the U.S.

Because patients in the Hepatitis C Antiviral Long-term Treatment against Cirrhosis Trial also had previously failed interferon therapy, it is not clear whether the results can be generalized to other patient populations. Future studies among patients with less advanced disease, those who are treatment-naïve to prior therapy, or who are being treated with newer antiviral agents are needed.

For more information on hepatitis, please read the AGA brochure "Understanding Hepatitis" at www.gastro.org/patient-center/digestive-conditions/hepatitis or visit the National Institute of Diabetes and Digestive and Kidney Diseases at http://digestive.niddk.nih.gov/ddiseases/pubs/hepc_ez.

http://www.eurekalert.org/pub_releases/2011-06/uomh-dsp060711.php

Drug shows promise in prostate cancer spread to bone

Bone scans show tumor shrinkage after Cabozantinib; bone pain reduced

ANN ARBOR, Mich. — A new drug to treat prostate cancer shows early promise, particularly against tumors that have spread to the bone, a multi-site study shows.

The drug Cabozantinib is designed to target mainly two important pathways linked to the growth and spread of prostate cancer. The drug had the most effect on tumors that had spread to the bone.

"Not only did three-quarters of bone scans have partial or complete resolution, but this was accompanied by improvement in bone pain and decreased need for narcotic use," says lead study author Maha Hussain, M.D.,

FACP, professor of internal medicine and urology and associate director of clinical research at the University Michigan Comprehensive Cancer Center.

Hussain presented the findings at the American Society of Clinical Oncology annual meeting.

The trial enrolled 171 men with metastatic prostate cancer. In more than three-quarters of the men enrolled, cancer had spread to the bone.

Researchers found 76 percent of patients saw some or all of their tumor shrink on bone scans following treatment with Cabozantinib. In addition, among patients who were on narcotics due to bone pain, 67 percent reported less pain and 56 percent either stopped taking narcotics or reduced the dosage. In addition, more than two-thirds of patients had some tumor regressions in areas of spread outside the bone. The treatment effects lasted on average 29 weeks. The study found moderate side effects from Cabozantinib, including fatigue, gastrointestinal symptoms and high blood pressure.

"What's interesting about this drug is it brings to the table something we haven't seen before. Dramatic improvements in bone scans are unprecedented in this disease. Despite measurable progress, current treatment options for advanced prostate cancer tend to be modest in effect, so adding to and improving these options is a high priority," Hussain says.

Hussain cautions that this is very early data, but it opens a new door for further investigation. The manufacturer, Exelixis, has developed a randomized clinical trial that is currently open at the U-M Comprehensive Cancer Center and other locations. For information, call the U-M Cancer AnswerLine at 800-865-1125.

U-M researchers are also planning a clinical trial with this drug in patients with metastatic prostate cancer who have had no previous chemotherapy. Laboratory research at the University of Michigan will look to better understand Cabozantinib's effects on the bone. Cabozantinib is not approved by the U.S. Food and Drug Administration.

Prostate cancer statistics: 217,730 Americans will be diagnosed with prostate cancer this year and 32,050 will die from the disease, according to the American Cancer Society

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http://www.eurekalert.org/pub_releases/2011-06/aeco-eoe060711.php

Einstein offers easy-to-use genome analyzer to scientific community

BRONX, NY – Scientists at Albert Einstein College of Medicine of Yeshiva University have developed a desktop genome analyzer and browser that allows biologists to rapidly and easily analyze and process their high-throughput data.

The open-source software, called GenPlay, is described in the May 19 online edition of Bioinformatics. Currently, genomic data is analyzed mainly by information specialists rather than by the biologists who designed the experiments that produce the data. GenPlay was created with the goal of offering biologists a user-friendly, multi-purpose tool that can help them visualize, analyze and transform their raw data into biologically relevant tracks.

"The first human genome was sequenced 10 years ago by an international consortium at a cost of \$7 billion," notes GenPlay co-developer Eric Bouhassira, Ph.D., senior author of the Bioinformatics article, professor of medicine and of cell biology, and the Ingeborg and Ira Leon Rennert Professor of Stem Cell Biology and Regenerative Medicine at Einstein. "But today, a complete genome can be sequenced for less than \$10,000 and the cost is predicted to drop to less than \$1,000 in a few years. The dramatic dip in cost has led to the creation of an avalanche of new data that biologists are having trouble analyzing. GenPlay is intended to make it easier for biologists to make sense of their data."

A dozen or so genome browsers are currently available. GenPlay offers a major advantage over the others, says Dr. Bouhassira, because it "emphasizes letting biologists take control of their own data by providing continuous visual feedback together with extremely rapid browsing at every decision point during an analysis."

GenPlay handles three major types of data: data from gene expression studies, epigenetic data, and single nucleotide polymorphism (SNP) data. The free GenPlay software is available from <http://www.genplay.net> (http://genplay.einstein.yu.edu/wiki/index.php/Main_Page).

The Bioinformatics paper is titled "GenPlay, a Multi-Purpose Genome Analyzer and Browser." The lead author of the paper is Julien Lajugie, M.S., associate in Einstein's department of medicine, who co-developed GenPlay and wrote the GenPlay program. The project was funded by New York State Stem Cell Science (NYSTEM) (<http://stemcell.ny.gov/>).

http://www.eurekalert.org/pub_releases/2011-06/tes-oad060711.php

Older age does not cause testosterone levels to decline in healthy men

A decline in testosterone levels as men grow older is likely the result—not the cause—of deteriorating general health, say Australian scientists, whose new study finds that age, in itself, has no effect on testosterone level in healthy older men.

The results, to be presented Tuesday at The Endocrine Society's 93rd Annual Meeting in Boston, are the first findings released from the Healthy Man Study, according to principal investigator David Handelsman, MD, PhD, professor and director of the ANZAC Research Institute at the University of Sydney.

"Some researchers believe that an age-related testosterone deficiency contributes to the deteriorating health of older men and causes nonspecific symptoms, such as tiredness and loss of libido," he said.

Handelsman and his team, however, found that serum (blood) testosterone levels did not decline with increasing age in older men who reported being in excellent health with no symptoms to complain of.

"We had originally expected age to have an effect on serum testosterone, so the findings were a bit of a surprise," Handelsman said.

Two study centers in Australia recruited 325 men over the age of 40 (median age, 60) who had self-reported excellent health and no symptom complaints. To test blood testosterone levels, the researchers took blood samples from the men nine times over three months. They excluded men from the study who took medications that affect testosterone. Obesity caused a mild and clinically unimportant lowering of blood testosterone levels, the investigators reported. Age had no effect on testosterone level.

"The modest decline in blood testosterone among older men, usually coupled with nonspecific symptoms, such as easy fatigue and low sexual desire, may be due to symptomatic disorders that accumulate during aging, including obesity and heart disease," he said. "It does not appear to be a hormone deficiency state."

The message for patients and their doctors, Handelsman said, is "older men with low testosterone levels do not need testosterone therapy unless they have diseases of their pituitary or testes."

This research was supported by the MBF (Medical Benefits Fund) Foundation in Sydney, which is part of the private health insurer Bupa.

http://www.eurekalert.org/pub_releases/2011-06/aaoo-bac060711.php

Bursitis a common cause of painful hips, knees, heels and elbows

Most conditions can be managed with simple, nonsurgical techniques

As warm weather arrives and the great outdoors beckons, more and more men and women will be taking to the trails, the beaches, or their yards and gardens, embarking on physical activities that may result in sore, aching, swollen joints.

While it may be tempting to ignore these aches and pains or treat them with a little over-the-counter liniment, a wiser choice is to visit a physician who can determine if the symptoms are due to bursitis, inflammation of the fluid-filled bursae, or sacs, that surround and cushion the joints.

Bursitis occurs when the bursae become irritated or infected, often causing pain on movement. When infection is involved, medical intervention is necessary to fight the underlying infection and prevent it from spreading; when infection is not involved, prompt medical attention can prevent the condition from becoming worse over time.

Four of the most common types of bursitis, affecting the hips (trochanteric bursitis), knees (prepatellar bursitis), elbows (olecranon bursitis) and heels (retrocalcaneal bursitis), are examined in a new review article published in the Journal of the American Academy of Orthopaedic Surgeons (JAAOS).

"Bursitis is a common cause of musculoskeletal pain and often prompts orthopaedic consultation," said study author Daniel Aaron, MD, a clinical instructor in the department of orthopaedics at Brown University in Providence, R.I. "One of the challenges facing clinicians is to differentiate bursitis from conditions with similar symptoms, including arthritis, tendinitis, fracture, tendon or ligament injury and tumor.

Additionally, bursitis arises from infectious and noninfectious causes, and distinguishing between the two can be challenging.

"A thorough history and physical examination is required for accurate diagnosis, and in some cases, medical tests also may be used to help the clinician identify bursitis and determine whether or not infection is involved," he added. Trauma or infection is usually the root cause of all four types of bursitis, Dr. Aaron said.

"Hip and heel bursitis usually result from 'overuse' syndromes involving underlying structures related to the tendons," he noted. "Elbow and knee bursitis can be traumatic, due to either chronic low-level trauma or acute trauma, or infectious. Other inflammatory conditions can lead to bursitis as well."

Typical symptoms of bursitis include:

- * pain with or without joint movement;
- * swelling of the area surrounding the joint;
- * redness of the skin near the joint;
- * warmth of the area near the joint; or
- * pain or tenderness when the bursa is touched.

Dr. Aaron noted not all types of bursitis will involve the same kinds of symptoms. For instance:

* Hip bursitis may involve pain on the side of the hip, often radiating to the thigh. The hip area may be painful to the touch. Although range of motion of the hip may appear normal during the physical exam, the symptoms of trochanteric bursitis may be exacerbated by lying on your side, walking (especially uphill), climbing stairs and standing up from a seated position.

* Knee bursitis may be due to specific predisposing factors, including a history of trauma to the area, such as repetitive or prolonged kneeling, immune system disorders, alcoholism, chronic obstructive pulmonary disease (COPD), kidney failure, prior use of local corticosteroid medication and previous inflammation of the bursa. Pain with movement is uncommon, except when the joint is significantly flexed.

* Elbow bursitis typically involves a history of minor or repetitive local trauma. Although swelling is often involved in patients with olecranon bursitis, usually this swollen area is only tender when infection is involved.

* Heel bursitis often involves pain surrounding the Achilles tendon and heel areas, which are often tender when squeezed. This type of bursitis is typically associated with overuse and is especially common in runners, especially those who regularly train on inclines.

All types of bursitis often can be successfully managed non-surgically, and possible treatments include:

- * use of ice packs or compressive dressings;
- * activity modification that may reduce stress or irritation;
- * administration of nonsteroidal anti-inflammatory drugs (NSAIDs) or antibiotics;
- * corticosteroid injections (knee and elbow);
- * stretching exercises; and/or
- * change of footwear (heel).

Surgery may be required in patients whose symptoms remain following these treatments and in certain situations when infection is involved.

Dr. Aaron said in most cases, the best way to prevent bursitis is to vary physical activity, avoiding repetitive activities that may increase stress and trauma on the joints. Padding surrounding the knee or elbow joints may help prevent repetitive trauma which could lead to bursitis in those areas. Finally, losing extra weight which may be causing stress on joints, particularly of the hips and knees, is also recommended. "By recognizing the presence of bursitis and determining whether or not infection is involved, clinicians can identify the best mode of treatment which will resolve symptoms and help the patient regain mobility," Dr. Aaron said.

<http://www.nytimes.com/2011/06/07/health/07really.html>

The Claim: Cranberry Juice Can Cure Ulcers.

By ANAHAD O'CONNOR

THE FACTS Cranberry juice has a long history as a home remedy for bladder infections. But scientists in recent years have quietly studied whether it might also work against Helicobacter pylori, the bacterium responsible for most ulcers.

Scientists have known for some time that the juice effectively prevents some species of bacteria from adhering to the cell receptors along the urinary tract, which in theory should reduce the risk of bladder infections. The same mechanism is believed to work against ulcer formation: Compounds in cranberry juice called proanthocyanidins are thought to keep H. pylori from adhering to the lining of the stomach.

Most studies have found that consuming cranberry juice does seem to produce improvement in people prone to ulcers. In one randomized, double-blind study published in the journal Nutrition in 2008, researchers followed 271 children and teenagers who tested positive for H. pylori.

Over three weeks, one group drank 200 milliliters of cranberry juice daily, another was given a probiotic supplement containing competing bacteria, and another received a placebo. At the end of the study, the cranberry group had significantly higher "eradication rates" of H. pylori than the placebo group, and a slightly better rate of improvement than the group taking only probiotics. A study of almost 200 people published in 2005 had similar results. Drinking one cup of cranberry juice daily eliminated H. pylori in three times as many subjects as a daily cranberrylike placebo juice, though some of the subjects experienced no benefit.

THE BOTTOM LINE Researchers have found that cranberry juice may help prevent ulcers.

Drugs hailed as a 'major breakthrough' in treating deadly skin cancer

Trials of vemurafenib and Yervoy deliver dramatic results in survival rates for those with metastatic melanoma, researchers say.

By Thomas H. Maugh II, Los Angeles Times

Two new drugs can significantly increase survival in patients with metastatic melanoma, the advanced and generally lethal form of skin cancer, researchers reported.

Results were so dramatic in a trial of one of the drugs that the study was halted early, researchers reported Sunday at a Chicago meeting of the American Society of Clinical Oncology. Studies on both drugs were published online by the New England Journal of Medicine.

Melanoma is among the most common cancers in the United States. An estimated 68,000 new cases are diagnosed each year, with 8,700 deaths.

The shortened trial is notable because the experimental agent called PLX4032, or vemurafenib, is the first chemotherapy agent directed at a specific mutation involved in the formation of skin tumors.

The drug's development "is a major defining moment that will have an important effect on survival and quality of life," wrote Dr. Marc S. Ernstoff of the Dartmouth Medical School in Lebanon, N.H., in an editorial accompanying the report. But the mutation that the drug targets occurs in only 47% of melanoma patients, and the drug appears to help only about half of them.

Many of the rest may benefit from the second drug, called ipilimumab and marketed as Yervoy, which stimulates the immune system to fight the tumors. Yervoy was approved by the Food and Drug Administration in March based on earlier results that showed it was more effective than a vaccine used to treat melanoma. The findings reported Sunday showed that it also was better than conventional chemotherapy.

The manufacturers of the two new drugs funded the studies and are planning trials to use the agents together to see if the combination can further improve outcomes.

The findings are "absolutely a major breakthrough for patients who have metastatic ... melanoma" or inoperable melanoma, said Dr. Sylvia Adams, a melanoma immunotherapy expert at the New York University School of Medicine and a spokeswoman for the American Society of Clinical Oncology. She was not involved in the studies.

The incidence of melanoma is increasing most rapidly among the elderly and among women ages 15 to 39, presumably because of excess exposure to ultraviolet radiation outdoors and in tanning salons.

The conventional treatment is with the drug dacarbazine, which produces a median survival of 5.6 to 7.8 months after treatment commences.

Vemurafenib targets the V600E mutation in a gene called BRAF, which is involved in cell growth. Small, early studies suggested the drug could be quite powerful.

In the new trial, a team headed by Dr. Paul Chapman of the Memorial Sloan-Kettering Cancer Center in New York enrolled 675 patients with the V600E mutation at 103 sites around the world. Half of the patients took dacarbazine, and half took vemurafenib.

After three months, those receiving vemurafenib had a 74% reduction in progression of the disease and a 63% reduction in the risk of death. About 48% of the patients' tumors showed a response to the drug, compared with only a 5.5% response in those taking dacarbazine.

The results were so powerful that the study's data-monitoring committee recommended halting the study and giving vemurafenib to all the patients.

Primary side effects were skin rashes, sensitivity to light and joint pain.

In the second study, a team led by Dr. Jedd Wolchok, also of Memorial Sloan-Kettering, enrolled 502 patients with metastatic melanoma who had never been treated. Half received Yervoy plus dacarbazine, and half received dacarbazine and a placebo.

After one year, 47.3% of patients who received the combination were still alive, compared with 36.3% of those receiving only dacarbazine. At two years, the figures were 28.5% for Yervoy and 17.9% in the control group, and after three years, 20.8% and 12.2%. The median survival for those taking Yervoy and dacarbazine was 11.2 months, compared with 9.1 months among those given only dacarbazine.

Side effects included elevated liver enzymes, diarrhea and rash.

The chief drawback of the drugs is cost. Yervoy, manufactured by Bristol-Myers Squibb, costs \$120,000 for a course of treatment. Roche and Plexxikon Inc., a biotechnology company in Berkeley that developed vemurafenib, are expected to apply to the FDA shortly for approval to market the drug but have not yet announced a price.

Spiders Evolved Spare Legs

Arachnids missing up to two limbs can build webs and hunt with ease.

Matt Kaplan for National Geographic News

Scientists may have uncovered why spiders are so creepy-crawly—they have more legs than they need, a new study says. After collecting thousands of female spiders in the wild, scientists found that more than 10 percent of the arachnids were missing at least one of their eight legs.

"We wondered if this was handicapping them in any way," said study co-author Alain Pasquet at the University of Nancy 1 in France.

The research team placed 123 *Zygiella x-notata* spiders in individual plastic boxes, where the animals could build webs. Sixty of the spiders were eight-legged, while 63 were each missing one or more legs.

Pasquet and his colleagues found that webs built by spiders missing at least one leg did not differ much from the webs built by spiders that were intact.

Six-Legged Spiders Still Good Hunters

The scientists then placed flies in the enclosures and found that leg-lacking spiders were also perfectly capable of catching and eating the insects. "We were really surprised—we expected missing a leg to harm the spiders' ability to catch food, and it didn't at all," Pasquet said.

Based on the findings, the authors propose that spiders have legs that they don't really need—an advantage when it comes to escaping a predator that's put the bite on a limb, for example.

Yet there does appear to be a limit to how many legs a spider can lose. In the wild, the team found few spiders missing more than two legs. And in the lab, these five-legged spiders built shoddy webs.

Spider-legs study published in a recent issue of the journal *Naturwissenschaften*.

http://www.eurekalert.org/pub_releases/2011-06/mu-lgi060811.php

Lifelong gap in health between rich and poor set by age 20

Study by McGill geography professor finds that as people age, the difference in the health-related quality of life between rich and poor remains constant

"We can't buy our way out of ageing," says Nancy Ross, a McGill geography professor. "As we get older we start to have vision problems, maybe some hearing loss, maybe lose some mobility – ageing is a kind of a social equalizer."

Ross is the lead author of a new study about how socio-economic and educational status affects Canadians' health-related quality of life over the course of a lifetime.

"My research looks at how poverty and social disadvantage affect your health status. Our work was about using social circumstances as a lens to look at how people's quality of life changes as they age."

The good news, according to Ross, is that there is no sign of an accelerated ageing process for those who are lower on the social ladder. "The trajectories for declining health as people age look fairly similar across the social spectrum. That surprised me. I thought that there would be a bit more of a difference across social groups."

But the bad news is that Canadians who are less educated and have a lower income start out less healthy than their wealthier and better-educated compatriots, and remain so over the course of their lives. "What we found, basically, is that people who are more educated and with higher incomes have a better health-related quality of life over their whole lifespan, and that these health "tracks" stay pretty parallel over time.

"The message there is that if you start out with a health-related quality of life deficit through early life experience and a poor educational background, it's never made up for later on," says Ross. "Poorer Canadians are in poorer health and they have lower life expectancy than their more affluent counterparts, and by age 20 the pattern for health-related quality of life as people age is already fixed."

"We might speculate that universal health insurance and other social policies directed to adults and seniors have played a role in preventing accelerated decline in health-related quality of life of the poorer and less educated Canadians. That said, we would need some comparative research in other countries to test this more fully," she adds. "But this study suggests the need for policies aimed at making sure kids and teens are given the chances early in life to even out socio-economic inequalities that will affect their health as they age."

The study analyzed data gathered from 17,000 Canadians who were questioned about their health seven times over a period of 16 years, between 1994/1995 and 2006/2007, by the National Population Health Survey.

The research was funded by the National Institute on Aging, the National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, and the Fonds de la recherche en santé du Québec.

To read the full study: <http://jech.bmj.com/content/early/2011/03/24/jech.2010.115378.long>

http://www.eurekalert.org/pub_releases/2011-06/tes-eah060811.php

Eating a high-fat diet may rapidly injure brain cells that control body weight

Obesity among people who eat a high-fat diet may involve injury to neurons, or nerve cells, in a key part of the brain that controls body weight, according to the authors of a new animal study.

"The possibility that brain injury may be a consequence of the overconsumption of a typical American diet offers a new explanation for why sustained weight loss is so difficult for most obese individuals to achieve," said presenting author Joshua Thaler, MD, PhD, a faculty member with the Diabetes and Obesity Center of Excellence at the University of Washington in Seattle. The results will be presented Tuesday at The Endocrine Society's 93rd Annual Meeting in Boston.

Thaler and his colleagues studied the brains of rodents for the short-term and long-term effects of eating a high-fat diet. After giving groups of six to 10 rats and mice a high-fat diet for periods from one day to eight months, the researchers performed detailed biochemical, imaging and cell sorting analyses on the animals' brains.

Within the first three days of consuming a diet that had a similar fat content to the typical American diet, rats consumed nearly double their usual daily amount of calories, Thaler reported. Rats and mice fed the high-fat diet gained weight throughout the study. These rodents developed inflammation in the hypothalamus, the part of the brain containing neurons that control body weight. At the same time, a group of support cells called glia and scavenger cells called microglia accumulated in the hypothalamus and appeared to become activated. Although this collective response to brain inflammation—called gliosis—subsided days later, it recurred after four weeks.

"Gliosis is thought to be the brain equivalent of wound healing and is typically seen in conditions of neuronal injury, such as stroke and multiple sclerosis," Thaler said. "We speculate that the early gliosis that we saw may be a protective response that fails over time."

In their experiments, Thaler said they also detected damage to, and eventual loss of, critical weight-regulating neurons. These neurons, called pro-opiomelanocortin (POMC) neurons, were reduced in number by month 8 of the high-fat diet in mice, according to Thaler. These results were not present in same-age rodents fed standard chow.

It is not yet clear whether this presumed neuronal injury is permanent, but it may contribute to weight gain, he stated.

This research, which was funded by the National Institute of Diabetes and Digestive and Kidney Diseases, provides a new potential target for obesity treatment, Thaler concluded.

"If new medicines can be designed that limit neuron injury during overeating, they may be effective in combating the obesity epidemic," he said.

http://www.eurekalert.org/pub_releases/2011-06/sp-lor060811.php

Lack of relationships, education top list of common American regrets

Los Angeles, CA Regrets—we've all had a few.

Although too many regrets can interfere with life and mental health, a healthy amount of regret can motivate us to improve our lives, say researchers Mike Morrison of the University of Illinois and Neal Roese of Northwestern University in the current issue of *Social Psychological and Personality Science* (published by SAGE).

The researchers telephoned a representative sample of nearly 400 Americans to ask them about what they regret. The most frequent regrets of Americans are about love, education, and work. Romantic regrets—America's most common—focused on lost chances for potential romances, and relationships that did not live up to their potential.

The other common regrets for Americans involved family, education, career, finances, and parenting. Women were more likely to have regrets about relationships (romance, family), and men were more likely to have regrets about work (career and education). It was the lack of romantic relationships and the lack of higher education that were regretted most.

At first, people tend to regret the things they've done more than the opportunities they didn't take. But with time, people come to regret more keenly the chances they let go past. Over their lives, Americans had more regret about the times they've lacked opportunities than about the choices they've made.

"We tend to regret matters that are most important to us," said Morrison, "people crave strong, stable social relationships and are unhappy when they lack them." Regret can be painful, but it can also be useful. "Some people say they try to live life without regret and, I think that's being unfair to the human condition," said Roese, "if we try to squeeze regrets away, we're sacrificing a bit of our humanity."

The article "Regrets of the Typical American: Findings From a Nationally Representative Sample" in Social Psychological and Personality Science, is available free for a limited time

http://www.eurekalert.org/pub_releases/2011-06/hu-ttm060811.php

Tut, tut: Microbial growth in pharaoh's tomb suggests burial was a rush job **A Harvard expert in cultural heritage microbiology, investigates a "fingerprint" left by ancient Egyptian microbes**

Cambridge, Mass - In the tomb of King Tutankhamen, the elaborately painted walls are covered with dark brown spots that mar the face of the goddess Hathor, the silvery-coated baboons—in fact, almost every surface.

Despite almost a century of scientific investigation, the precise identity of these spots remains a mystery, but Harvard microbiologist Ralph Mitchell thinks they have a tale to tell.

Nobody knows why Tutankhamen, the famed "boy king" of the 18th Egyptian dynasty, died in his late teens. Various investigations have attributed his early demise to a head injury, an infected broken leg, malaria, sickle-cell anemia, or perhaps a combination of several misfortunes.

Whatever the cause of King Tut's death, Mitchell thinks those brown spots reveal something: that the young pharaoh was buried in an unusual hurry, before the walls of the tomb were even dry. Like many ancient sites, Tutankhamen's tomb suffers from peeling paint and cracking walls. In the oppressive heat and humidity, throngs of tourists stream in and out of the cave, admiring it but also potentially threatening it.

Concerned about the tomb's preservation, the Egyptian Supreme Council of Antiquities approached the Getty Conservation Institute for help. The Getty, in turn, had questions for Mitchell. What are the brown spots? Are visiting tourists making them worse? Most importantly, do they present a health hazard?

In his investigation, Mitchell, the Gordon McKay Research Professor of Applied Biology at Harvard's School of Engineering and Applied Sciences (SEAS), combines classical microbiology with cutting-edge genomic techniques. His research team has been culturing living specimens swabbed from the walls of the tomb as well as conducting DNA sequence analyses.

Meanwhile, chemists at the Getty have been analyzing the brown marks, which have seeped into the paint and the plaster, at the molecular level. So far, the chemists have identified melanins, which are characteristic byproducts of fungal (and sometimes bacterial) metabolism, but no living organisms have yet been matched to the spots.

"Our results indicate that the microbes that caused the spots are dead," says Archana Vasnathakumar, a postdoctoral fellow in Mitchell's lab. "Or, to put it in a more conservative way, 'not active.'"

Further, analysis of photographs taken when the tomb was first opened in 1922 shows that the brown spots have not changed in the past 89 years.

While the identity of the ancient organism remains a mystery, all of this is good news for tourists and Egyptologists alike, because the evidence suggests that not only are the microbes not growing—they're actually part of the history, offering new clues to the circumstances of King Tut's death.

"King Tutankhamen died young, and we think that the tomb was prepared in a hurry," explains Mitchell. "We're guessing that the painted wall was not dry when the tomb was sealed."

That moisture, along with the food, the mummy, and the incense in the tomb, would have provided a bountiful environment for microbial growth, he says, until the tomb eventually dried out.

Exotic as the project may sound, investigations like this are typical of Mitchell's research in applied microbiology. In past years, his lab has studied the role of bacteria in the deterioration of the USS Arizona at Pearl Harbor, Hawaii, and the microorganisms living within limestone at Mayan archaeological sites in southern Mexico. Nick Konkol, a former postdoctoral research associate, and Alice DeAraujo, a current research assistant, have developed rapid new ways to detect mold growing within the paper of historical manuscripts, paintings, and museum artifacts.

The field is referred to as "cultural heritage microbiology," and Mitchell literally wrote the textbook on it.

For microbiologists with broad interests, cultural heritage provides an endless supply of surprising, new applications, crossing disciplines and cultures and providing important insight into modern environmental problems.

"This type of research is typical of the interactive activity of SEAS, where modern scientific and engineering techniques are integrated to solve complex problems," Mitchell says.

Just a few years ago, he was called down to the Smithsonian National Air and Space Museum to investigate the collection of Apollo space suits. In the heat and humidity of the museum's Maryland storage facility, black mold was chewing through the many-layered polymers, damaging the priceless suits.

The relatively simple solution in that case was the installation of a climate control system. Unfortunately, however, there is a difference between prevention and treatment. Once a historical artifact has begun to deteriorate, the damage is usually irreversible.

Mitchell points to the example of the cathedral in Cologne, Germany. Built over the course of 632 years and listed as a UNESCO World Heritage site, the walls of the magnificent cathedral feature angels and historical figures carved out of stone. In just the past 100 years, the angels' faces have been eaten away by air pollution.

"I always use the analogy of cancer," Mitchell says. "You want to get to it early enough that it isn't doing major destruction."

But what to do about King Tut's 3000-year-old microbial vandalism?

The damage is already done, so Mitchell predicts that the conservators will want to leave the spots alone, particularly as they are unique to that site. "This is part of the whole mystique of the tomb," he says.

http://www.eurekalert.org/pub_releases/2011-06/cshl-asv060611.php

Autism study validates importance of spontaneous causal mutations and sheds new light on gender skew

Genetic causation of ASD appears to be highly diverse; Thoughts on why fewer girls have autism

Cold Spring Harbor, NY – A clinically extensive and mathematically powerful study of 1000 families with one autistic child and one unaffected sibling has validated a controversial theory of autism's complex genetic causation. The study for the first time estimates the minimum number of locations in the human genome -- 250 to 300 -- where gene copy number variation (CNV) can give rise to autism spectrum disorder (ASD). It also sheds new light on the long observed but little understood "gender bias" of autism, an illness that typically manifests by age 3 and affects about four times more boys than girls.

The study, along with an accompanying paper exploring the possible brain and neuronal pathologies to which the observed CNVs and other genetic anomalies may give rise, appears June 9 in the journal *Neuron*. The work was led by Professor Michael Wigler of Cold Spring Harbor Laboratory (CHSL), a pioneer in the analysis of genomes and a developer of key technologies that have made such analysis possible, in collaboration with Dennis Vitkup of Columbia University; Dan Levy, Michael Ronemus, Ivan Iossifov and Sarah Gilman of CSHL; and others.

"The causes of autism when fully fleshed out are likely to be very diverse," Wigler says, "some of which may be treatable much more readily than others. However, the diversity of causes implies that an effective future treatment for one form of ASD may be specific only for a narrow subset of those affected."

Four years ago, Wigler and colleagues began to publish findings about the genetics of autism that surprised many experts in the field. Among other things, they noted the prevalence of "de novo" genetic mutations in affected children. These are mutations that did not appear in either parent and hence must have arisen spontaneously. In 2008, Wigler proposed that such cases, characterized by changes in gene copy number -- duplicated or deleted genome segments that leave an individual with extra or missing copies of one or more genes -- likely account for at least half of ASD cases.

There is little doubt that certain genetic mutations carried by an unaffected parent can be transmitted to a child and cause the illness. Wigler, however, proposed a "unified theory" of autism genetics that suggested such cases only account for about 25% of the total.

Rare and ultra-rare CNVs – and their possible biological consequences

The new study affirms the team's earlier data on the relative importance of non-inherited, spontaneously occurring CNVs as likely causal factors in autism. Using a newly assembled sample population called the Simons Simplex Collection which included about 1000 families, consisting of both birth parents and in most cases two children, one with ASD and the other unaffected, Wigler and colleagues saw the earlier-observed effect, only "this time on a much larger scale." The new sample collection is deemed very important by the research team. It intentionally excludes families with more than one child with ASD ("multiplex families") since such cases are almost always inherited. A sample composed only of "simplex" families makes possible a much more accurate comparison of the relative frequency of spontaneous and inherited causal mutations.

Using a refined version of a technique called comparative genomic hybridization, but using detection devices called microarrays that were about four times as powerful as those used in prior studies, along with a suite of powerful quantitative analysis tools, the team was able to consistently resolve much smaller genomic irregularities than previously possible. This revealed a significantly greater number of genome areas where spontaneous DNA deletions and duplications "hit" genes thought to contribute substantially to ASD.

The team estimates a minimum of 250 to 300 places in the genome where CNVs give rise to ASD. Most of the CNVs were only seen once and are classified, individually, as rare. This is a potential source of confusion since these rare events, taken in total, are the source of at least half of all autism, if Wigler's theory is correct.

As the team notes in their paper, it is still very difficult to make the connection between the areas that the de novo CNVs hit and the genes these areas harbor. Nor is it clear what role those genes have in perturbing the biology of the developing brain, giving rise to autism. In the associated paper, Vitkup, Iossifov and others use a novel form of analysis called NETBAG (network-based analysis of genetic associations) to identify "the large biological network of genes affected by rare de novo CNVs in autism." They note that this network is strongly related to genes previously implicated in studies of autism and intellectual disability. And they specify they relate primarily to the development of synapses, the junctions between brain cells which are their transmission nodes; as well as the targeting of axonal fibers and the ability of young neurons to migrate. All are essential aspects of brain development.

Explaining gender skew and apparent female 'resistance' to autism

Subsequent sequence-based studies should be able to further resolve individual genes within the CNV regions, and significantly expand the list of candidate genes for autism. Even now, however, the data on spontaneous CNVs and a separate but also an important class of "ultra-rare" CNVs that were inherited by some of the affected children in the sample, reveal interesting and in some cases vexing "asymmetries," the team reported.

Among these asymmetries: spontaneous causal CNVs were found in 8% of the affected children in the sample; this is four times their rate of occurrence in unaffected siblings. Since CNVs occur with equal frequency across the human population – we all have them, but in most of us the missing or extra genetic material doesn't hit essential genes or contribute to illness – the fact that they are seen four times as often in affected children as in their unaffected siblings points to their likely contributory role in the illness.

Another striking asymmetry: when spontaneous CNVs contributing to autism were seen in girls, they tended to be much larger and hit many more genes -- 15 genes per CNV on average for girls vs. only 2 for boys. This touches on three observed phenomena: the preponderance of boys who become symptomatic; the preponderance of boys who have ASD but are "high-functioning"; and the comparative severity of the illness when it does occur in girls.

Since girls and boys are equally susceptible to DNA damage and new mutation -- the rate of new mutation that damages a gene in humans is about one gene per three births -- they should each have about the same number of CNVs. Presuming this is the case, why do more boys than girls develop ASD? And why are the observed CNVs in girls larger?

Wigler hypothesizes that females are in some manner "resistant" to autism, and that it takes a much larger, more devastating genomic hit to induce pathology in girls as compared with boys. The cause of this apparent female resistance to comparatively small genetic hits remains one of autism's mysteries.

It has been speculated that unequal rates of brain development in the sexes may account for the smaller number of females with ASD. Wigler's team discusses this possibility, noting that females "have an accelerated timescale for a number of cognitive milestones, for example, their first words at an earlier age. A quicker pace of development might reflect a robustness that offers females protection."

Another mystery concerning gender draws attention to the status of adult women of child-bearing age who might be considered "carriers." These would be women whose genomes contain transmissible mutations that can cause autism, yet who do not themselves manifest symptoms of ASD. The team speculates that the "high-risk" autism genes they carry may generate other effects in these women. They "may encounter difficulties at later stages of their lives that manifest as a different diagnostic category, or one that reduces fecundity." If this is true, the disorder is "most likely to be one with a gender bias opposite that of ASD," such as anorexia nervosa, Wigler proposes.

The team's next insights about autism genetics will be made possible by a major technological advance -- a shift from microarray detection of CNVs to the sequencing of the gene-encoding portions of the genome, or "exome," of families in the Simons Simplex Collection (which will expand to include 3000 families). This work and work at other institutions of a similar nature should "crack open the field of autism genetics in two years," says Wigler, whose own team has been gearing up for the sequencing phase during the last year.

"Rare de novo and transmitted copy number variation in autistic spectrum disorders" will be published June 9 in Neuron. The authors are: Dan Levy, Michael Ronemus, Boris Yamron, Yoon-ha Lee, Anthony Leotta, Jude Kendall, Steven Marks, B. Lakshmi, Deepa Pai, Kenny Ye, Andreas Buja, Abba Krieger, Seungtae Yoon, Jennifer Troge, Linda Rodgers, Ivan Iossifov and Michael Wigler.

"Rare de novo variants associated with autism implicate a large functional network of genes involved in formation and function of synapses" will be published June 9 in Neuron. The authors are: Sarah R. Gilman, Ivan Iossifov, Dan Levy, Michael Ronemus, Michael Wigler and Dennis Vitkup.

http://www.eurekalert.org/pub_releases/2011-06/yu-yrd060611.php

Yale researchers discover many genetic keys needed to unlock autism

Hundreds of small genetic variations are associated with autism spectrum disorders, including an area of DNA that may be a key to understanding why humans are social animals, according to a multi-site collaborative study led by researchers at Yale University.

Published in the June 9 issue of the journal *Neuron*, the study reinforces the theory that autism, a disorder that develops in early childhood involving impairments in social interaction, language deficits and distinctive behaviors, is not caused by one or two major genetic defects, but by many small variations, each associated with a small percentage of cases.

The study—led by Matthew State, M.D., Ph.D., the Donald J. Cohen Associate Professor of Child Psychiatry, Psychiatry and Genetics—looked at more than 1,000 families in which there was a single child with an autism spectrum disorder, an unaffected sibling and unaffected parents. The team, including postdoctoral fellow and first author Stephan Sanders from Yale, compared individuals with autism to their siblings to determine what types of genetic changes distinguished the affected child from the unaffected child.

"Thanks to an ambitious collaboration among a large group of autism researchers from around the country, supported by the Simons Foundation, we were able to focus on an ideal study population," said State, who is co-director of the Yale Program on Neurogenetics. "It made all the difference in our ability to identify several regions of the genome clearly contributing to autism."

One of the most intriguing of these findings points to the same small section of the genome that causes Williams syndrome—a developmental disorder marked by high sociability and an unusual aptitude for music.

In autism, there is an increase in the chromosomal material, an extra copy of this region, and in Williams syndrome, there's a loss of that same material," said State. "What makes this observation particularly interesting is that Williams syndrome is known for a personality type that is highly empathetic, social, and sensitive to the emotional state of others. Individuals with autism often have difficulties in the opposite direction. This suggests that there is an important key in that region to understanding the nature of the social brain."

State and his team also found about 30 other regions in the genome that are very likely contributing to autism and are focused on about six of those regions that showed the strongest evidence. "We're now moving on to a second phase of the study looking at an additional 1,600 families and should be able to identify multiple new regions that are strongly implicated in autism," he said.

Sanders and State are optimistic about the new findings, pointing out that genetics is the first step to understanding what's really going on at the molecular and cellular level of the brain. "We can use these genetic findings to begin unraveling the underlying biology of autism," said Sanders. "This will help tremendously in the effort to identify new and better approaches to treatment."

Two other studies published in the June 9 issue of *Neuron* report on the same families studied by State, Sanders and their co-authors. One of these, by a group at Cold Spring Harbor Laboratory in New York, paints a very similar picture—that autism is a highly genetically diverse disorder and that sporadic changes in the structure of the genome present only in the affected individuals and not in other families often play a key role. The other study, by researchers at Columbia University, suggests that although hundreds of genes may be involved in autism, they appear to disrupt a common molecular network involved in the mobility of brain cells and development of synapses between them.

Additional Yale authors on the study include Gulhan Ercan-Sencicek, Michael Murtha, Abha Gupta, Kaya Bilguvar, Murim Choi, Nicole Davis Wright, Nicholas DiLullo, Thomas Fernandez, Gerald Goh, Kyle Meyer, John Murdoch, Gordon Ober, Melanie Raubeson, Youeun Song, Murat Günel, Richard Lifton and Shrikant Mane.

The following authors participated in the study at Yale but are now at other institutions: Christopher Mason, Rahul Dhodapkar, Vikram Fielding-Singh, Daniel Fishman, Sindhuja Kammela, Brian O'Roak, Rebecca Pottenger and Ilana Yurkiewicz. Citation: Neuron, Vol. 70, Issue 5 (June 9, 2011).

http://www.eurekalert.org/pub_releases/2011-06/idso-sfs060811.php

Study finds shingles may be related to elevated risk of multiple sclerosis

Taiwanese investigators have found that there can be a significantly higher risk of multiple sclerosis (MS) occurring in the year following a shingles, or herpes zoster, attack.

The findings, which support a long-held view on how MS may develop, are published in *The Journal of Infectious Diseases* and now available online (<http://jid.oxfordjournals.org/content/early/2011/06/07/infdis.jir239.abstract>).

MS is an autoimmune disease that affects the brain and spinal cord, leading to inflammation and nerve damage as the body's immune cells attack the nervous system. Possible causes that may trigger the

inflammation include environmental, genetic, and viral factors. One virus that has been associated with MS is varicella zoster virus, the cause of herpes zoster.

In a study conducted by Herng-Ching Lin, PhD, and colleagues at Taipei Medical University in Taiwan, 315,550 adults with herpes zoster and a control group of 946,650 subjects were tracked and then evaluated for MS occurrence during a one-year follow-up period. The control group was selected randomly from a pool of subjects who had not been diagnosed with herpes zoster or other viral diseases. After adjusting for monthly income and geographic region, the authors found that the group with herpes zoster had a 3.96 times higher risk of developing MS than the control group. The authors noted that this risk, although increased, was still low, as is the frequency of MS in general. The study also noted an interval of approximately 100 days between a herpes zoster event and occurrence of MS.

Although the study was limited almost entirely to Han Chinese adults, the large scope of this nationwide case-controlled study, 1.26 million sampled patients, provides strong epidemiological evidence for a possible role for herpes zoster in the development of MS. The authors also point out that MS has a lower prevalence in Asian compared to Western populations and, thus, it may be difficult to project their findings to other populations.

In an accompanying editorial (<http://jid.oxfordjournals.org/content/early/2011/06/07/infdis.jir243.extract>), Teresa Corona, MD, and Jose Flores, MD, of the National Institute of Neurology and Neurosurgery in Mexico noted that "The evidence provided in this study...allows us to better understand the role of these viral factors as an MS risk among certain genetically susceptible individuals," and that the study should be corroborated in other parts of the world to help clarify the role of this and other viruses in MS.

Fast Facts:

1. There is epidemiological evidence that some herpes viruses may contribute to multiple sclerosis (MS) occurrence.
2. The rate of MS prevalence varies by geographical location and income.
3. In this study, investigators found a significantly higher—but still low—risk for MS occurring in the year following a shingles, or herpes zoster, attack compared to a control population.
4. There is evidence that 30 percent of relapses in MS patients may be associated with an infectious disease.

<http://medicalxpress.com/news/2011-06-blamed-aging-ros-molecules-life.html>

Once blamed for aging, ROS molecules may actually extend life

(Medical Xpress) -- In a new study, Yale University researchers have identified a pathway by which reactive oxygen species (ROS) molecules, which are usually implicated in the aging process due to their damage to DNA, can also act as cellular signaling molecules that extend lifespan.

The study, which provides insights into the underlying mechanisms of the ROS signaling process, is published in the June issue of *Cell Metabolism*.

Increased ROS, and their effects at the cellular level, can lead to oxidative stress, which is involved in many diseases and aging. But ROS are also necessary for the proper functioning of the immune system and other biological functions. Using the model organism yeast, the Yale team set out to determine whether regulating ROS and their ability to act as signaling molecules could impact the aging process.

Inhibiting a signaling pathway called Target of Rapamycin (TOR), which is involved in sensing nutrients and cell growth, increases lifespan in yeast, as it does in mice. The Yale team found that a key way this occurs is by altering the function of cellular powerhouses called mitochondria so that they produce more signaling ROS.

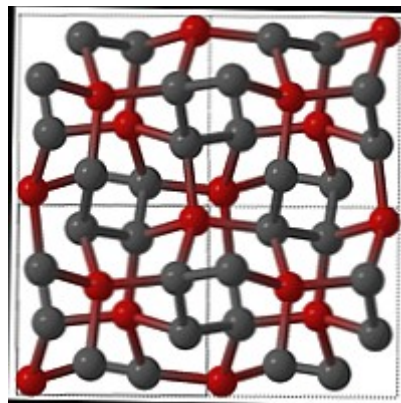
"The concept that ROS are important cellular signaling molecules, and not just agents of damage and stress, has grown to be widely accepted," said lead author Gerald S. Shadel, Ph.D., professor of pathology and genetics at Yale School of Medicine. "Remarkably, in this study, we show that their purposeful production by mitochondria can even provide an adaptive signal that can delay aging."

Since the TOR pathway operates largely the same in yeast as it does in humans, the new connections to mitochondrial ROS signaling and aging in this study may be more widely applicable. Shadel said that new ways to intervene in age-related pathology may stem from these basic studies. "Trials targeting the TOR pathway as an anti-cancer strategy in humans are already underway. Our study suggests that carefully augmenting mitochondria and ROS production in humans may also be beneficial in combating aging and associated diseases."

Other authors are Yong Pan and Elizabeth A. Schroeder of Yale School of Medicine; Alejandro Ocampo of the Yale Department of Biochemistry and Molecular Biology; and Antonio Barrientos of Yale and the University of Miami Miller School of Medicine. Provided by Yale University

Researchers predict material 'denser than diamond' *Researchers have predicted three new forms of carbon*

(PhysOrg.com) -- Stony Brook University graduate student Qiang Zhu, together with Professor of Geosciences and Physics, Artem R. Oganov, postdoc Andriy O. Lyakhov and their colleagues from the University de Oviedo in Spain, have predicted three new forms of carbon, the findings of which were published in a paper entitled "Denser than diamond: Ab initio search for superdense carbon allotropes," in the June 7, 2011 online edition of Physical Review B. So far, each new found modification of carbon resulted in a scientific, technological revolution – the same could happen now, if scientists can find a way to synthesize these new forms of carbon.



Elemental carbon possesses a unique range of structures and properties – from ultrsoft graphite to superhard diamond, and also including elusive carbines, beautifully symmetric fullerenes, carbon nanotubes, and the recently established new form, M-carbon (the structure of which was predicted by Oganov in 2006). Properties of all these modifications of carbon are so interesting and so tunable that two Nobel prizes were awarded recently for their studies (the 1996 Chemistry and 2010 Physics awards).

Graphene is the densest two-dimensional material, with unique mechanical and electronic properties and having some electrons moving with near-light velocities and behaving as if they had zero mass. Diamond has set several records – it is not only the hardest known material, but also has denser packing of atoms than any other known three-dimensional material. When doped by boron, diamond displays superconductivity and is the only known materials simultaneously displaying superhardness and superconductivity.

Now Zhu, Oganov, and their colleagues propose three new structures of carbon, which should be more than 3% denser than diamond. Greater density means that electrons should have a higher kinetic energy (that is, move faster). Calculations of Zhu et al. show that the new modifications are almost as hard as diamond, but do not exceed its hardness. Their electronic properties are very diverse, with the band gap ranging from 3.0 eV to 7.3 eV. Band gap is the minimum separation in energy between occupied and unoccupied electronic orbitals and is the most important characteristic of the electronic structure of materials. Such a wide range of band gaps implies the possibility of tuning the electronic properties. The band gap of 7.3 eV predicted for the tP12 modification is the largest value for all forms of carbon.

Other interesting properties include ultralow compressibility – when subjected to pressure, the new forms of carbon will contract less than most materials (even slightly less than diamond, the current record holder). They have higher refractive indices and stronger light dispersion than diamond – which means better brilliance and color effects than those displayed by diamond. “Carbon is an inexhaustible element in its chemical diversity and in the multitude of its physical applications”, says Professor Oganov. “If these predicted forms of carbon can be synthesized, they may find important technological roles”. Researchers believe that the new forms of carbon, thanks to their high densities, could be synthesized by shock compression of low-density modifications, or by directed growth on substrate.

*More information: Denser than diamond: Ab initio search for superdense carbon allotropes, Phys. Rev. B 83, 193410 (2011)
DOI:10.1103/PhysRevB.83.193410*

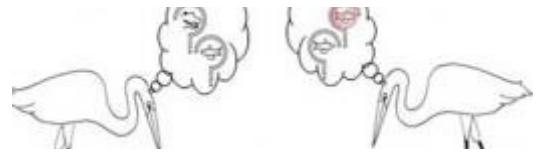
Abstract

Diamond has the highest number density (i.e., the number of atoms per unit volume) of all known substances and a remarkably high valence electron density ($n_{\text{val}} = 0.697 \text{ \AA}^{-3}$). Searching for possible superdense carbon allotropes, we have found three structures (hP3, tI12, and tP12) that have significantly greater density. The hP3 and tP12 phases have strong analogy with two polymorphs of silica (β -quartz and keatite), while the tI12 phase is related to the high-pressure SiS₂ polymorph. Furthermore, we found a collection of other superdense structures based on the motifs of the aforementioned structures, but with different ways of packing carbon tetrahedra, and among these the hP3 and tI12 structures are the densest. At ambient conditions, the hP3 phase is a semiconductor with the GW band gap of 3.0 eV, tI12 is an insulator with the band gap of 5.5 eV, while tP12 is an insulator, the band gap of which is remarkably high (7.3 eV), making it the widest-gap carbon allotrope. These allotropes are metastable and have comparable to diamond or slightly higher bulk moduli; their Vickers hardnesses are calculated to be 87.6 GPa for hP3, 87.2 GPa for tI12, and 88.3 GPa for tP12, respectively, thus making these allotropes nearly as hard as diamond (for which the same model gives the hardness of 94.3 GPa). Superdense carbon allotropes are predicted to have remarkably high refractive indices and strong dispersion of light. Provided by Stony Brook University

Learn to pay attention

(Medical Xpress) -- A new scientific theory on what we learn to pay attention to and what we learn to ignore could turn 30 years of research on its head.

Research by Dr Mark Haselgrove from The University of Nottingham (UK), and Dr Guillem Esber from the University of Maryland (USA), challenges two long held and contradictory theories on which cues our brains use to predict events of significance.



The theory, published today (Wednesday June 8 2011) in the journal Proceedings of the Royal Society B, has important implications for the psychology and neuroscience of attention. It has implications for our understanding of how uncertainty, such as the uncertainty surrounding a risky investment, may bias our attention. Furthermore, by advancing the theoretical basis of how learning influences attention, this research may inform our understanding of what happens when the allocation of attention is inappropriate, such as occurs in mental illnesses such as schizophrenia.

Dr Haselgrove said: "Animals, and that includes humans, spend a great deal of their waking hours learning about and using cues to predict events of significance — such as food, danger, or the opportunity to have sex. One question that has long captivated the imagination of psychologists is how animals come to attend to the appropriate cues. Decades of research have singled out two variables — predictiveness and uncertainty — as key factors in determining how much attention animals and humans pay to a cue."

It turns out that existing theories that have tried to explain the influence of these two variables on attention are contradictory. One theory suggests attention is captured by cues that are good predictors of significant events — to enable animals to work out what cues are relevant to them. The rival theory argues instead that attention is applied where it is most needed — to cues that may or may not be followed by events of significance — that is to say, to cues that possess uncertainty. Both of these theories seem intuitively plausible and have scientific evidence to back them up but they are, unfortunately, contradictory.

"Can the brain really be wired up to attend to the world in two contradictory ways?" asks Dr Haselgrove. "Surely there must be a resolution to this problem."

Dr Esber said: "The basis for solving the problem is to appreciate that uncertainty can be thought of as another type of predictiveness. For example, the ripples on a lake caused by a fish under its surface may frequently help a hungry heron in his hunt and will be associated with a tasty meal. However, the fish will sometimes be too quick for the heron, or the ripples will be caused by the wind — and the heron will go hungry. Although the ripples can be thought of as an uncertain cue for fish, they are in fact predictive of two things: the satisfaction associated with a tasty meal, and the frustration that accompanies hunger."

In other words, uncertainty is a situation where a cue is predictive of two opposite events.

From this insight Dr Haselgrove and Dr Esber were able to apply the principles of associative learning that have developed since the time of Pavlov's investigations into conditioned reflexes to devise an entirely new explanation for how learning influences attention in animals and importantly resolve the contradiction between uncertainty and predictiveness. *Provided by University of Nottingham*

<http://www.bbc.co.uk/news/health-13699711>

Drug makes hearts repair themselves

By James Gallagher Health reporter, BBC News

The damage caused by a heart attack had previously been considered permanent.

But a study in the journal Nature showed the drug, thymosin beta 4, if used in advance of a heart attack, was able to "prime" the heart for repair. The British Heart Foundation described repair as the "holy grail of heart research", but said any treatment in humans was years away.

Due to advances in health care the number of people dying from coronary heart disease is falling. But those living with heart failure are on the rise - more than 750,000 people have the condition in the UK alone.

Wake up

The researchers at University College London looked at a group of cells which are able to transform into different types of heart tissue in an embryo.

Professor Paul Riley, from the University College London, said: "The adult epicardial cells which line the muscle of the heart can be activated, move inward and give rise to new heart muscle."

"We saw an improvement in the ejection fraction, in the ability of the heart to pump out blood, of 25%."

As well as pumping more blood, the scar tissue was reduced and the walls of the heart became thicker.

Peter Weissberg, medical director of the British Heart Foundation, said he was "very excited" about the research but warned the scale of improvement seen in animals was rarely seen in humans.

Heart Epicardium derived progenitor cells (in red) lining the heart

However, he argued that even a small improvement would have a dramatic impact on people's quality of life.

"A normal heart has lots of spare capacity. In patients with heart failure it is working flat out just to sit down [and] it's like running a marathon," he said. "You could turn a patient from somebody who's gasping while sitting in a chair to somebody who can sit comfortably in a chair."

Advance therapy

The mice needed to take the drug in advance of a heart attack in order for it to be effective. As the researchers put it, "the priming effect is key". If a similar drug could be found to be effective in humans, then the researchers believe it would need to be prescribed in a similar way to statins.

Professor Riley said "I could envisage a patient known to be at risk of a heart attack - either because of family history or warning signs spotted by their GP - taking an oral tablet, which would prime their heart so that if they had a heart attack the damage could be repaired." He said this could be available in 10 years.

The British Heart Foundation, which funded the study, said repairing a damaged heart was the "holy grail" of heart research. The results strengthened the evidence that drugs could be used to prevent the onset of heart failure, it said.

<http://www.bbc.co.uk/news/world-africa-13688683>

Hedge funds 'grabbing land' in Africa

Hedge funds are behind "land grabs" in Africa to boost their profits in the food and biofuel sectors, a US think-tank says.

In a report, the Oakland Institute said hedge funds and other foreign firms had acquired large swathes of African land, often without proper contracts. It said the acquisitions had displaced millions of small farmers.

Foreign firms farm the land to consolidate their hold over global food markets, the report said. They also use land to "make room" for export commodities such as biofuels and cut flowers. "This is creating insecurity in the global food system that could be a much bigger threat than terrorism," the report said.

The Oakland Institute said it released its findings after studying land deals in Ethiopia, Tanzania, South Sudan, Sierra Leone, Mali and Mozambique. It said hedge funds and other speculators had, in 2009 alone, bought or leased nearly 60m hectares of land in Africa - an area the size of France.

'Risky manoeuvre'

When I visited Lungi-Lol in rural Sierra Leone I saw men hoeing thousands of hectares of farmland owned by Addax, a Swiss-based bio-energy company. They are growing sugarcane to produce biofuels.

Campaigners say this contributes to food insecurity, but many people here welcome Addax's presence.

Francis Koroma, who works on the farm, says: "We thank God for Addax. I am gainfully employed and I receive about \$70 (£46) a month. Before, I spent a whole year without getting \$50."

Villagers are unaware of the controversy surrounding biofuels.

Abdulai Conteh, a local traditional leader, said: "Some people are doing business here but I have no idea what they are doing with our land. I see them growing sugarcane. That's all I know. The same financial firms that drove us into a global recession by inflating the real estate bubble through risky financial manoeuvres are now doing the same with the world's food supply," the report said. It added that some firms obtained land after deals with gullible traditional leaders or corrupt government officials.

"The research exposed investors who said it is easy to make a deal - that they could usually get what they wanted in exchange for giving a poor tribal chief a bottle of Johnnie Walker [whisky]," said Anuradha Mittal, executive director of the Oakland Institute. "When these investors promise progress and jobs to local chiefs it sounds great, but they don't deliver."

The report said the contracts also gave investors a range of incentives, from unlimited water rights to tax waivers. "No-one should believe that these investors are there to feed starving Africans.

"These deals only lead to dollars in the pockets of corrupt leaders and foreign investors," said Obang Metho of Solidarity Movement for New Ethiopia, a US-based campaign group.

However, not all companies named in the report accept that their motives are as suggested and they dismiss claims that their presence in Africa is harmful. One company, EmVest Asset Management, strongly denied that it was involved in exploitative or illegal practices.

"There are no shady deals. We acquire all land in terms of legal tender," EmVest's Africa director Anthony Poorter told the BBC. He said that in Mozambique the company's employees earned salaries 40% higher than the minimum wage. The company was also involved in development projects such as the supply of clean water to rural communities. "They are extremely happy with us," Mr Poorter said.

A new way to make lighter, stronger steel -- in a flash

COLUMBUS, Ohio – A Detroit entrepreneur surprised university engineers here recently, when he invented a heat-treatment that makes steel 7 percent stronger than any steel on record – in less than 10 seconds.

In fact, the steel, now trademarked as Flash Bainite, has tested stronger and more shock-absorbing than the most common titanium alloys used by industry.

Now the entrepreneur is working with researchers at Ohio State University to better understand the science behind the new treatment, called flash processing. What they've discovered may hold the key to making cars and military vehicles lighter, stronger, and more fuel-efficient.

In the current issue of the journal *Materials Science and Technology*, the inventor and his Ohio State partners describe how rapidly heating and cooling steel sheets changes the microstructure inside the alloy to make it stronger and less brittle.

The basic process of heat-treating steel has changed little in the modern age, and engineer Suresh Babu is one of few researchers worldwide who still study how to tune the properties of steel in detail. He's an associate professor of materials science and engineering at Ohio State, and Director of the National Science Foundation (NSF) Center for Integrative Materials Joining for Energy Applications, headquartered at the university.

"Steel is what we would call a 'mature technology.' We'd like to think we know most everything about it," he said. "If someone invented a way to strengthen the strongest steels even a few percent, that would be a big deal. But 7 percent? That's huge." Yet, when inventor Gary Cola initially approached him, Babu didn't know what to think. "The process that Gary described – it shouldn't have worked," he said. "I didn't believe him. So he took my students and me to Detroit."

Cola showed them his proprietary lab setup at SFP Works, LLC., where rollers carried steel sheets through flames as hot as 1100 degrees Celsius and then into a cooling liquid bath.

Though the typical temperature and length of time for hardening varies by industry, most steels are heat-treated at around 900 degrees Celsius for a few hours. Others are heated at similar temperatures for days.

Cola's entire process took less than 10 seconds.

He claimed that the resulting steel was 7 percent stronger than martensitic advanced high-strength steel. [Martensitic steel is so named because the internal microstructure is entirely composed of a crystal form called martensite.] Cola further claimed that his steel could be drawn – that is, thinned and lengthened – 30 percent more than martensitic steels without losing its enhanced strength.

If that were true, then Cola's steel could enable carmakers to build frames that are up to 30 percent thinner and lighter without compromising safety. Or, it could reinforce an armored vehicle without weighing it down.

"We asked for a few samples to test, and it turned out that everything he said was true," said Ohio State graduate student Tapasvi Lolla. "Then it was up to us to understand what was happening."

Cola is a self-taught metallurgist, and he wanted help from Babu and his team to reveal the physics behind the process – to understand it in detail so that he could find ways to adapt it and even improve it.

He partnered with Ohio State to provide research support for Brian Hanhold, who was an undergraduate student at the time, and Lolla, who subsequently earned his master's degree working out the answer.

Using an electron microscope, they discovered that Cola's process did indeed form martensite microstructure inside the steel. But they also saw another form called bainite microstructure, scattered with carbon-rich compounds called carbides.

In traditional, slow heat treatments, steel's initial microstructure always dissolves into a homogeneous phase called austenite at peak temperature, Babu explained. But as the steel cools rapidly from this high temperature, all of the austenite normally transforms into martensite.

"We think that, because this new process is so fast with rapid heating and cooling, the carbides don't get a chance to dissolve completely within austenite at high temperature, so they remain in the steel and make this unique microstructure containing bainite, martensite and carbides," Babu said.

Lolla pointed out that this unique microstructure boosts ductility -- meaning that the steel can crumple a great deal before breaking – making it a potential impact-absorber for automotive applications.

Babu, Lolla, Ohio State research scientist Boian Alexandrov, and Cola co-authored the paper with Badri Narayanan, a doctoral student in materials science and engineering.

Now Hanhold is working to carry over his lessons into welding engineering, where he hopes to solve the problem of heat-induced weakening during welding. High-strength steel often weakens just outside the weld joint, where the alloy has been heated and cooled. Hanhold suspects that bringing the speed of Cola's method to welding might minimize the damage to adjacent areas and reduce the weakening.

If he succeeds, his discovery will benefit industrial partners of the NSF Center for Integrative Materials Joining Science for Energy Applications, which formed earlier this year. Ohio State's academic partners on the center include Lehigh University, the University of Wisconsin-Madison, and the Colorado School of Mines. http://www.eurekalert.org/pub_releases/2011-06/jhmi-wad060911.php

Why animals don't have infrared vision

Johns Hopkins researchers uncover the source of the visual system's 'false alarms'

On rare occasion, the light-sensing photoreceptor cells in the eye misfire and signal to the brain as if they have captured photons, when in reality they haven't. For years this phenomenon remained a mystery. Reporting in the June 10 issue of *Science*, neuroscientists at the Johns Hopkins University School of Medicine have discovered that a light-capturing pigment molecule in photoreceptors can be triggered by heat, as well, giving rise to these false alarms.

"A photon, the unit of light, is just energy, which, when captured by the pigment rhodopsin, most of the time causes the molecule to change shape, then triggering the cell to send an electrical signal to the brain to inform about light absorption," explains King-Wai Yau, Ph.D., professor of neuroscience at Johns Hopkins and member of its Center for Sensory Biology. "If rhodopsin can be triggered by light energy," says Yau, "it may also be occasionally triggered by other types of energy, such as heat, producing false alarms. These fake signals compromise our ability to see objects on a moonless night. So we tried to figure it out; namely, how the pigment is tripped by accident."

"Thermal energy is everywhere, as long as the temperature is above absolute zero," says neuroscience research associate Dong-Gen Luo, Ph.D. "The question is: How much heat energy would it take to trigger rhodopsin and enable it to fire off a signal, even without capturing light?" says Johns Hopkins Biochemistry, Cellular and Molecular Biology graduate student Wendy Yue.

For 30 years, the assumption was that heat could trigger a pigment molecule to send a false signal, but through a mechanism different from that of light, says Yau, because it seemed, based on theoretical calculations: that very little thermal energy was required compared to light energy.

But the theory, according to Yau, was based mainly on the pigment rhodopsin. However, rhodopsin is mainly responsible for seeing in dim light and is not the only pigment in the eye; other pigments are present in red-, green- and blue-sensitive cone photoreceptors that are used for color and bright-light vision. Although researchers are able to measure the false events of rhodopsin from a single rhodopsin-containing cell, a long-standing challenge has been to take measurements of the other pigments. "The electrical signal from a single cone pigment molecule is so small in a cone cell that it is simply not measurable," says Luo. "So we had to figure out a new way to measure these false signals from cone pigments."

By engineering a rod cell to make human red cone pigment, which is usually only found in cone cells, Yau's team was able to measure the electrical output from an individual cell and calculate this pigment's false signals by taking advantage of the large and detectable signals sent out from the cell.

As for blue cone pigment, "Nature did the experiment for us," says Yau. "In many amphibians, one type of rod cells called green rods naturally express a blue cone pigment, as do blue cones." So to determine whether heat can cause pigment cells to misfire, the team, working in the dark, first cooled the cells, and then slowly returned the cells to room temperature, measuring the electrical activity of the cells as they warmed up. They found that red-sensing pigment triggers false alarms most frequently, rhodopsin (bluish-green-sensing pigment) triggers falsely less frequently, and blue-sensing pigment does so even less.

"This validates the 60-year-old Barlow's hypothesis that suggested the longer wavelength the pigment senses—meaning the closer to the red end of the spectrum—the noisier it is," says Yau. And this finding led the team to develop and test a new theory: that heat can trigger pigments to misfire, by the same mechanism as light.

Pivotal to this theory is that visual pigment molecules are large, complex molecules containing many chemical bonds. And since each chemical bond has the potential to contain some small amount of thermal energy, the total amount of energy a pigment molecule could contain can, in theory, be enough to trigger the false alarm.

"For a long time, people assumed that light and heat had to trigger via different mechanisms, but now we think that both types of energy, in fact, trigger identical changes in the pigment molecules," says Yau. Moreover, since longer wavelength pigments have higher rates of false alarms, Yau says this may explain why animals never evolved to have infrared-sensing pigments.

"Apart from putting to rest a long-standing debate, it's a wake-up call for researchers to realize that biomolecules in general have more potential thermal energy than previously thought," says Luo.

This study was funded by the National Institutes of Health, the Antonio Champalimaud Vision Award and The Academy of Finland.

Asteroid served up 'custom orders' of life's ingredients

Some asteroids may have been like "molecular factories" cranking out life's ingredients and shipping them to Earth via meteorite impacts, according to scientists who've made discoveries of molecules essential for life in material from certain kinds of asteroids and comets.

Now it appears that at least one may have been less like a rigid assembly line and more like a flexible diner that doesn't mind making changes to the menu.

In January, 2000, a large meteoroid exploded in the atmosphere over northern British Columbia, Canada, and rained fragments across the frozen surface of Tagish Lake. Because many people witnessed the fireball, pieces were collected within days and kept preserved in their frozen state. This ensured that there was very little contamination from terrestrial life.

"The Tagish Lake meteorite fell on a frozen lake in the middle of winter and was collected in a way to make it the best preserved meteorite in the world," said Dr. Christopher Herd of the University of Alberta, Edmonton, Canada, lead author of a paper about the analysis of the meteorite fragments published June 10 in the journal *Science*.

"The first Tagish Lake samples -- the ones we used in our study that were collected within days of the fall -- are the closest we have to an asteroid sample return mission in terms of cleanliness," adds Dr. Michael Callahan of NASA's Goddard Space Flight Center in Greenbelt, Md., a co-author on the paper.

The Tagish Lake meteorites are rich in carbon and, like other meteorites of this type, the team discovered the fragments contained an assortment of organic matter including amino acids, which are the building blocks of proteins. Proteins are used by life to build structures like hair and nails, and to speed up or regulate chemical reactions. What's new is that the team found different pieces had greatly differing amounts of amino acids.

"We see that some pieces have 10 to 100 times the amount of specific amino acids than other pieces," said Dr. Daniel Glavin of NASA Goddard, also a co-author on the *Science* paper. "We've never seen this kind of variability from a single parent asteroid before. Only one other meteorite fall, called Almahata Sitta, matches Tagish Lake in terms of diversity, but it came from an asteroid that appears to be a mash-up of many different asteroids."

By identifying the different minerals present in each fragment, the team was able to see how much each had been altered by water. They found that various fragments had been exposed to different amounts of water, and suggest that water alteration may account for the diversity in amino acid production.

"Our research provides new insights into the role that water plays in the modification of pre-biotic molecules on asteroids," said Herd. "Our results provide perhaps the first clear evidence that water percolating through the asteroid parent body caused some molecules to be formed and others destroyed. The Tagish Lake meteorite provides a unique window into what was happening to organic molecules on asteroids four-and-a-half billion years ago, and the pre-biotic chemistry involved."

If the variability in Tagish Lake turns out to be common, it shows researchers have to be careful in deciding whether meteorites delivered enough bio-molecules to help jump-start life, according to the team.

"Biochemical reactions are concentration dependent," says Callahan. "If you're below the limit, you're toast, but if you're above it, you're OK. One meteorite might have levels below the limit, but the diversity in Tagish Lake shows that collecting just one fragment might not be enough to get the whole story."

Although the meteorites were the most pristine ever recovered, there is still some chance of contamination though contact with the air and surface. However, in one fragment, the amino acid abundances were high enough to show they were made in space by analyzing their isotopes.

Isotopes are versions of an element with different masses; for example, carbon 13 is a heavier, and less common, variety of carbon. Since the chemistry of life prefers lighter isotopes, amino acids enriched in the heavier carbon 13 were likely created in space.

"We found that the amino acids in a fragment of Tagish Lake were enriched in carbon 13, indicating they were probably created by non-biological processes in the parent asteroid," said Dr. Jamie Elsila of NASA Goddard, a co-author on the paper who performed the isotopic analysis.

The team consulted researchers at the Goddard Astrobiology Analytical Lab for their expertise with the difficult analysis. "We specialize in extraterrestrial amino acid and organic matter analysis," said Dr. Jason Dworkin, a co-author on the paper who leads the Goddard laboratory. "We have top-flight, extremely sensitive equipment and the meticulous techniques necessary to make such precise measurements. We plan to refine our

techniques with additional challenging assignments so we can apply them to the OSIRIS-REx asteroid sample return mission."

OSIRIS-REx (Origins, Spectral Interpretation, Resource Identification, Security -- Regolith Explorer) is a Goddard-managed mission, led by the University of Arizona, that will be launched toward asteroid "1999 RQ36" in 2016 and return a sample to Earth in 2023. The OSIRIS-REx team is led by Dr. Michael Drake, Director of the University of Arizona's Lunar and Planetary Laboratory.

The Tagish Lake research was funded by the Natural Sciences and Engineering Research Council of Canada, the Alberta Ingenuity Fund, and NASA.

http://www.eurekalert.org/pub_releases/2011-06/w-sut060911.php

Sucking up to the boss may move you up and keep you healthy

Ingratiation used by politically savvy individuals neutralizes psychological distress

Savvy career minded individuals have known for some time that ingratiating oneself to the boss and others – perhaps more commonly known as 'sucking up'– can help move them up the corporate ladder more quickly. However, a recent study published in the Journal of Management Studies suggests that politically savvy professionals who use ingratiation as a career aid may also avoid the psychological distress that comes to others who are less cunning about their workplace behavior.

This new research shows that when politically savvy professionals use the coping skill of ingratiation, they may neutralize ostracism and other psychological distress that other less savvy individuals have to cope with in the workplace. Ostracized employees experience more job tension, emotional exhaustion and depressed mood at work.

Workplace ostracism—an adult form of bullying—is often described as an individual's belief that they are ignored or excluded by superiors or colleagues in the workplace. A 2005 survey of 262 full-time employees found that over a five-year period, 66% of respondents felt they were systematically ignored by colleagues, and 29% reported that other people intentionally left the area when they entered. Previous studies have shown that ostracism is an interpersonal stressor that can lead to psychological distress, and distress in the workplace is strongly linked to life distress, employee turnover, and poor physical health.

In the present study, researchers examined the relationship between workplace ostracism and employee psychological distress, with a focus on moderating effects of ingratiation and political skill. The research team surveyed employees from two oil and gas companies in China, with 215 employees providing responses. "Our data confirmed that workplace ostracism was positively related to psychological distress," explains Ho Kwong Kwan one of the study's authors. "We found that ingratiation neutralized the relationship between workplace ostracism and psychological distress when used by employees with a high level of political skill, but exacerbated the association when ingratiation was used by employees with low political savvy."

While the path to success and health may appear to come from sucking up, the authors of the study have a better suggestion. They say that organizations should create a culture that discourages workplace ostracism by provide training to managers and employees, which enhances self-esteem, encourages effective problem solving techniques, and promotes the development of political skills.

Full Citation "Coping with Workplace Ostracism: The Roles of Ingratiation and Political Skill in Employee Psychological Distress." Long-Zeng Wu, Frederick Hong-kit Yim, Ho Kwong Kwan, Xiaomeng Zhang. *Journal of Management Studies*; Published Online: June 8, 2011 (DOI: 10.1111/j.1467-6486.2011.01017.x). <http://onlinelibrary.wiley.com/doi/10.1111/j.1467-6486.2011.01017.x/abstract>

<http://news.discovery.com/human/psychic-fiasco-texas-mass-murder-raid-a-hoax-110609.html>

Psychic Fiasco: Texas Mass Murder Raid a Hoax

By Benjamin Radford | Thu Jun 9, 2011 10:24 AM ET

A psychic called police on Monday night, describing a horrific scene of mass murder: 25 to 30 dismembered bodies near an unassuming ranch house about an hour outside of Houston, Texas. There were rotting limbs, headless corpses, and, chillingly, many were children.

Deputies from the Liberty County Sheriff's office went to investigate but didn't see anything amiss.

The psychic called a second time the next day, insisting that her visions were true. She provided more detailed information about the home and urged the police to return to a different part of the property. This time detectives called for backup and soon dozens of officials from the Texas Department of Public Safety, the FBI, and the Texas Rangers were on the scene -- not to mention cadaver dogs, news helicopters, and gawkers.

Police investigated, and it all turned out to be a false alarm. There were no dead bodies; the psychic was wrong (or lying). How could one anonymous psychic have been taken so seriously by the police, FBI, and Texas Rangers? According to an Associated Press story:

Sheriff's Capt. Rex Evans said the fact that the tip came from someone claiming to be a psychic did not make it any less valid. Although his agency had never acted on a tip from a psychic, the department has received valid anonymous tips that resulted in arrests.

"I do believe there was perhaps a moment of pause, yes," Evans said. "However, due to the severity of the allegations she was making, we felt it prudent to at least make the attempt to investigate it and to determine whether or not the information she had given had any validity or not."

This case highlights the difficulty that police have in dealing with psychics. Law enforcement officials are in a tough spot: They cannot arbitrarily ignore credible information about a crime -- especially one that may involve dozens of dead children. Whether the tip comes from an ordinary citizen, an anonymous informant, or a self-proclaimed psychic, the information might be true.

So the police weren't "fooled," instead they were doing their jobs. The fault lies not with law enforcement but with the psychic who misled them, though police should of course approach psychic information with a great deal of skepticism.

This is only the most recent of many false psychic tips and predictions. In March 2004, a Florida psychic contacted the Transportation Safety Administration to inform them that a bomb was aboard an American Airlines flight headed for Dallas, Texas.

Nothing was found, but the delay caused by the psychic's tip forced cancellation of the flight, and over 100 passengers were placed on later flights, most delayed until the following day. Doug Perkins, local administrator for the TSA, echoed Sheriff Evans, saying: "We can't ignore anything."

While this incident is making national news, the scenario is actually fairly common. Police do not use psychics in investigations, though psychics often give unsolicited tips, leads and information -- especially in high profile cases. Hundreds of psychics -- some of them nationally known, such as Sylvia Browne, Carla Baron, Allison Dubois, and Noreen Renier -- have offered information on missing persons, yet not a single one has been recovered using their information.

There are countless examples of false information provided by psychics. But what about the successes, the cases where psychics have led police to find missing persons? There are none. Zero.

Despite popular belief and claims to the contrary, there is not a single documented case of a missing person being found or recovered due to psychic information. Psychics have consistently failed to find missing persons, including high-profile disappearances like Natalee Holloway and Holly Bobo.

And it's worth noting that psychics had nearly a decade to find Osama bin Laden, and spectacularly failed. It would take only one psychic with accurate, specific, verifiable information to save innocent lives, prove that psychics are real, and collect the \$27 million reward.

Psychic information often wastes police time and resources following up on false leads. In perhaps the most glaring and tragic failure of psychics, Jaycee Dugard, the girl who was abducted and held hostage by a California husband and wife, was subjected to continual sexual and physical abuse for nearly 20 years while psychics offered wrong and useless information about her location and condition.

Some may argue that the woman who sparked the Texas search isn't really psychic, but only pretending to be. The problem, of course, is that since psychic abilities have never been proven to exist, there's little meaningful distinction between a person who knows she is not psychic and a person who thinks that she is.

It's not as if the "real" psychics can do anything that the "fake" psychics can't. Anyone can give the police wrong information.

<http://www.newscientist.com/article/dn20558-cheap-vaccine-eradicates-new-cases-of-meningitis-a.html>

Cheap vaccine eradicates new cases of meningitis A

*** 16:01 09 June 2011 by Andy Coghlan**

It has taken just six months for a cheap new vaccine against meningitis A to work its magic: reducing the number of new cases in three west African countries to almost zero.

The outcome is a huge boost for a part of the world where meningitis A accounts for 90 per cent of all meningitis cases and where epidemics periodically kill tens of thousands. In 2009, between 6000 and 8000 people died and 55,000 contracted the disease in Nigeria alone. "It's all looking very promising," says Marie-Pierre Preziosi of the World Health Organization, head of a long-term project to roll out the new vaccine, dubbed MenAfriVac, to all countries in Africa's notorious "meningitis belt", which stretches through 25 countries from Senegal to Sudan. So far, no recipient of the vaccine has been infected, and the few cases that have occurred in treated areas were unvaccinated visitors from neighbouring areas.

The most comprehensive programme is in Burkina Faso, a landlocked country in west Africa where 20 million individuals aged 1 to 29 received the jab last year. Preziosi says there have been no cases of meningitis A in those vaccinated, compared with the usual 100 to 200 cases expected in six months, even when there is no

epidemic. A similar picture emerged in neighbouring Mali and Niger, which six months ago vaccinated people in zones considered most at risk for meningitis A, as a prelude to population-wide vaccinations.

Cheap and potent

MenAfriVac is much cheaper than existing meningitis A vaccines, at 50¢ compared with \$120 per dose. It is also more potent. Unlike conventional vaccines, which are based on sugars resembling those on the surface of *Neisseria meningitidis*, a bacterium that causes meningitis, the new vaccine splices the sugars to a carrier protein that is better at stirring up the body's immune system. "It makes the immune response much more vigorous," says Marc LaForce, director of the global Meningitis Vaccine Project, which developed MenAfriVac.

Antibodies against the bacterium continue to be produced long after vaccination, providing hope that a single jab may be enough to give lifelong protection. Nevertheless, it will be a long haul extending the vaccine to all countries at risk. Later this year, vaccinations will begin in Cameroon, Chad and Nigeria.

In an analysis based on the early results, LaForce and Jean-Marie Okwo-Bele of the WHO estimate that giving the vaccine in just seven countries could prevent a million cases over a decade and save \$300 million in the costs of treatment and lost ability to work. Many people who survive the disease are left disfigured or disabled, and need long-term care and treatment: brain damage occurs in 20 per cent of cases, for example.

They warn that much more money, an additional \$475 million, is needed to extend the jabs to between 250 and 300 million children and young adults in all 25 countries within the meningitis belt over the next five or six years. *Journal reference: Health Affairs, DOI: 10.1377/hlthaff.2011.0328*

<http://news.discovery.com/animals/dogs-are-likely-born-with-canine-telepathy-110609.html>

Dogs Likely Born with 'Canine Telepathy'

By Jennifer Viegas | Thu Jun 9, 2011 01:41 PM ET

Dogs are so in tune with us that they can read our minds, according to a new Learning & Behavior study that also determined canines are probably born with the ability.

Practice makes perfect, however, so the more a dog hangs around humans, the better he or she becomes at "canine telepathy," which actually relies upon hyperawareness of the senses.

Those of us who have owned or been around dogs for any period of time know how well they often "get" us, sensing tiredness, depression, headaches or other maladies before we consciously exhibit any major outward signs of distress. Dogs can even detect when a person has cancer. They also seem to sense our joy and good health.

Monique Udell and her team from the University of Florida wondered why dogs are so clever at reading us, and how they accomplish this feat. Are dogs born with the ability to sense our mental states, or do canines learn from experience?

To explore these questions and more, Udell and her team carried out two experiments involving both wolves and dogs. In the experiments, the two sets of animals were given the opportunity to beg for food, either from an attentive person or from a person unable to see the potential begger.

The researchers showed for the first time that wolves, like domestic dogs, are capable of begging successfully for food by approaching the attentive human. This demonstrates that both species - domesticated and non-domesticated - have the capacity to behave in accordance with a human's attentional state. They are therefore likely born with the ability, since wolves would not have had much practice, which the typical pet dog gains by begging for treats during dinner and at other times.

Some dogs were better at reading people than others were, however. Shelter dogs were not nearly as good as pampered house pooches, demonstrating that exposure to humans allows dogs to hone their natural people-reading skills more.

According to the researchers, "These results suggest that dogs' ability to follow human actions stems from a willingness to accept humans as social companions, combined with conditioning to follow the limbs and actions of humans to acquire reinforcement. The type of attentional cues, the context in which the command is presented, and previous experience are all important."

<http://news.nationalgeographic.com/news/2011/06/110609-coelacanths-long-lived-fish-science-animals/>

Coelacanths Can Live Past 100, Don't Show Age?

Two-decade study unlocks secrets of "living fossil" fish.

Matt Kaplan for National Geographic News

Not only is the coelacanth one of the world's oldest fish species, but the individual fish may also be long-lived.

A new study suggests the ancient fish can live up to a hundred years and even longer.

Until 1938, when a coelacanth (pronounced SEE-la-kanth) was found off Africa's coast, scientists had believed the fish went extinct 65 million years ago with a related lineage of prehistoric fishes.

After the coelacanth's rediscovery, a number of populations were uncovered in parts of the western Indian Ocean and in the western Pacific Ocean. Whether these populations were interconnected was a mystery.

"People kept catching these fish, but that didn't tell us anything about their population, how numerous they were, or if they were maybe simply strays from different parts of the ocean," said study leader Hans Fricke, an ethologist—or expert in animal behavior—formerly of the Max Planck Institute in Bremen, Germany.

Coelacanths Studied via Submersible

This lack of reliable data led Fricke and colleagues to begin a 21-year study of a coelacanth population found near the Comoros, a group of islands between the Seychelles and Madagascar. Because the fish live at depths of about 525 to 650 feet (160 to 200 meters), sending divers down to observe the fish was out of the question.



A coelacanth near South Africa's Sodwana Bay. Photograph by Laurent Ballesta, National Geographic

Instead, the team used submersibles to photograph, videotape, and study the fish. Because coelacanths have unique white markings on their sides, the team was able to identify more than 140 individuals during hundreds of submersible trips. But the team couldn't find any youngsters in the population of 300 to 400 coelacanths.

There's also little known about how the fish are born, noted Fricke, whose study appeared recently in the journal *Marine Biology*.

"We darted a pregnant female with a pinger [a type of tracking device] and followed her descent into the deep, so we think mothers may be going to great depths to give birth," he speculated.

Even stranger, only three or four coelacanths seemed to die each year, and their places in the population were taken by three or four new adults that would just mysteriously show up from nowhere.

Because roughly 4.4 percent of a given population of coelacanths appear to die each year—a figure at the lower end of observed mortality rates among fish—Fricke estimated that coelacanths have a longevity of about 103. Other fish, such as deepwater rockfishes of the genus *Sebastes*, have similar death rates and live for about a hundred years. With so few deaths and so few replacements in the population, Fricke argues that the evidence is clear that these fish are very long-lived.

Youthful-Looking Fish Hard to Age

Even so, the fish don't show the ravages of time, which makes determining their age very hard.

What's more, normal methods for measuring fish ages, such as measuring growth rings on their scales, aren't possible with coelacanths. That's because coelacanth scales don't seem to change over time like other fish, Fricke said. "We photographed some adults that arrived at the colony in 1989, and they did not grow at all. You just can't look at a coelacanth and speculate about age."

<http://www.physorg.com/news/2011-06-humans-extinction-proof.html>

Are humans extinction-proof?

Darren Curnoe

Does climate change seriously threaten to wipe out the human species if left unchecked?

Examining our evolutionary past suggests it might once have been the perfect catalyst for our extinction. But now?

On January 14 of this year, the Bulletin of the Atomic Scientists moved the hands of its Doomsday Clock one minute further from midnight (it's now six minutes to midnight), encouraged, it was announced, by the "progress seen around globe in both key threat areas: nuclear weapons and climate change".

First published in 1947, the bulletin was founded by scientists, engineers and other experts involved in the Manhattan Project. The clock continues to serve as a metaphorical countdown to the apocalypse – the annihilation of humanity – set for midnight.

Today, the bulletin's Board of Sponsors, comprising no less than 18 Nobel Laureates, almost every one of them a physicist or chemist, sets the hands of the clock based on their reading of "threats to the survival and development of humanity from nuclear weapons, climate change, and emerging technologies in the life sciences". They've a much wider brief now, a longer list of threats, and, I guess, more reasons to be pessimistic.

Around 500 million years ago, animal life was almost non-existent on Earth. Today, biologists recognise up to 6 million animal species.

Humanity – *Homo sapiens* – is just one among the 4,500 living mammal species; and some understanding of where we might be headed can be gleaned from where we've been – our evolutionary journey.

Our starting point as a group of two-footed, small-toothed, weakly-muscled, brainy “have-a-chat” apes is the ancestor we share with living chimpanzees some 7 million years ago. (The two chimpanzee species are endangered, incidentally, because of the environmental destruction caused by us, their closest cousin).

Our evolutionary group – the hominins – diversified quickly after the split from the human-chimp ancestor, and through its multiple evolutionary iterations natural selection produced 25 or 30 two-footed ape species – undoubtedly with more to be found as anthropologists discover more fossils. All of these are now extinct, except us.

Those 7 million years represent only the last couple of minutes on a 24-hour clock of Earth’s 5 billion year history. The culling of 30 species to 1 in this short timeframe, or a more than 95% loss of hominin biodiversity, is worse than the worst mass extinction episode recorded in the fossil record: the Permian event some 250 million years ago. But these mass events obscure the fact that, in the history of life, extinction has been a dominant theme, a continuous process. Evidence from the last 600 million years shows roughly one-third of existing animal species going extinct every 10 million years.

Seen in this context, the rate of extinction in the human evolutionary tree is striking, about three times faster than normal. This strongly suggests that we hominins are a highly extinction-prone mammal.

Why the dramatic loss of hominin diversity? What caused all these species to disappear? These are difficult and complex questions, but the answer may in part centre on the dramatic changes in climate that provided the backdrop for much of our evolution.

The last half million years or so in particular represent an episode of especially severe climate fluctuation, with intensely cold periods followed by warm phases, flip-flopping between the two on timescales of hundreds or thousands of years – in short, the worst bit of the 2.6 million-year Ice Age or Pleistocene Epoch.

The archaeological record of Europe suggests that vast areas were largely emptied of hominins during cold phases only to be recolonised during warm periods.

Hominins, pre-dating our own species, were living in Europe at latitudes as high as 53° north by 700,000 years ago. The 53rd parallel runs from the United Kingdom east through the Netherlands, Germany, Poland, Belarus, Russia, Kazakhstan, China (Inner Mongolia), United States (Alaska), Canada and Ireland.

Many places at this latitude today experience temperatures as low as -40° Celsius. But the climate at that time was Mediterranean in character. Soon after, the planet plunged into another cold phase lasting 100,000 years, with vast areas of Europe covered by ice.

Biologists have identified various intrinsic features of mammal species that increase their chances of extinction. They include traits such as:

- large body size;
- narrow ecological breadth (i.e. specialist feeders);
- low abundance, or sparse numbers of individuals, in the landscape as well as fluctuation in population over time.

Hominins are large mammals. Estimates of mass and stature for many Ice Age species would easily qualify them for spots on the front row of a rugby team ... and that’s just the females!

Large mammals are slow to mature and reproduce, and normally have one offspring at a time. While many extinct hominins were, like our own species, omnivorous, those living in cold climates relied heavily on animal food, as have recent hunter-gatherers such as the Inuit. This represents a narrowing of dietary niche on a par with many carnivores.

Estimates of population size from this period are remarkably low, with perhaps only 5,000 individuals in warm phases, plummeting to 1,000 or less during the cold stages, probably for the whole of Europe.

If around today, these individuals would be part of an endangered species, vulnerable to rapid extinction. And all of this applied to our own species as well for all but the last little bit of our brief evolutionary history.

Around 10,000 years ago, something unprecedented occurred that altered the course of our evolution: we invented farming. This massive change in dietary, social and economic behavior, a cultural shift known as the Neolithic Revolution, shaped the future course of our own, and the planet’s, evolution in remarkable and unpredictable ways. It resulted in anatomical, physiological and genetic changes that massively altered our evolution. Our domestication of plants and animals, and the large-scale clearing of land, altered the history of many others as well. It paved the way for a rise in infectious disease, and social changes such as occupational specialisation, writing, standing armies and empires, long distance trade, money and markets.

But the most profound shift of all was an explosion in human population, the result of greatly improved food security resulting in a dramatic lowering of infant and childhood mortality. In Europe, from a base of perhaps

only 5,000 Ice Age hunter-gatherers, the take-up of farming from approximately 8,000 years ago sharply increased population growth to an estimated rate of 3% per annum, from a long-term average of zero.

This is roughly three times today's global annual growth rate. From a population of less than 100,000 people worldwide, we have grown in less than 10,000 years to almost 7 billion.

Seen in its broadest context, the history of life on Earth soberly demonstrates that the vast majority of organisms that ever lived, perhaps 99% of them, no longer do. It also shows that mammal species normally last 1-2 million years before extinction inevitably bumps them off. Yet, unlike most mammals, including our dozens of extinct hominin cousins, we have escaped the vulnerabilities of a small and massively fluctuating population.

The simple, but profound act, of growing our own food delivered us the food security that ensured most of our children survived and our population grew. In effect, farming gave our species level assurance that the biological isn't always inevitable. The odds have shifted to such a degree that we may now be, with or without climate change, extinction-proof.

More information: This article was originally published by [The Conversation](#).

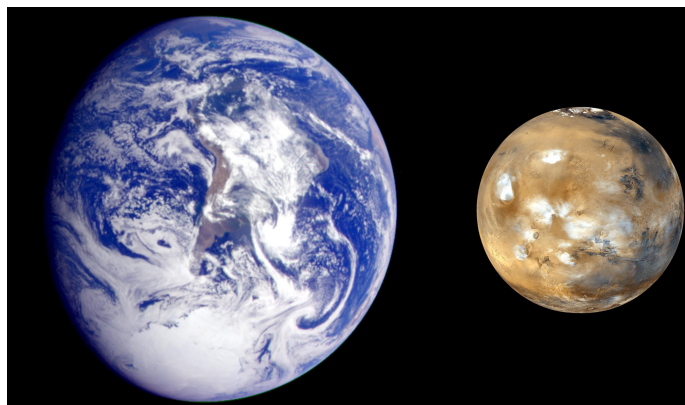
<http://www.scientificamerican.com/blog/post.cfm?id=what-would-happen-if-earth-and-mars-2011-06-09>

What Would Happen If Earth and Mars Switched Places?

By George Musser | Thursday, June 9, 2011

Imagine what would happen to our solar system if you suddenly swapped Earth and Mars

Last Saturday, at a workshop organized by the Foundation Questions Institute, Nobel laureate physicist Gerard 't Hooft gave a few informal remarks on the deep nature of reality. Searching for an analogy to the symmetries of basic physics, he asked the attendees to imagine what would happen to our solar system if you suddenly swapped Earth and Mars. He went on to discuss his ideas for explaining quantum mechanics, but couldn't get my mind off his question. What would happen?



Obviously, Martians would be delighted with the new arrangement. A fairly modest increase in Mars's

temperature would melt the polar caps and liberate gases from the soil, flipping the Martian climate into a new, cozier state nearly as warm as Earth. In an article for us in 1999, planetary scientist Chris McKay envisioned terraforming Mars by building factories to pump out greenhouse gases—proving that one man's poison is another's elixir—but moving the planet closer to the sun would certainly do the trick, too. Earthlings would get the short end of the deal. Sunlight would be half as intense and the planet would freeze over. On the plus side, we'd instantly be half as many years old.

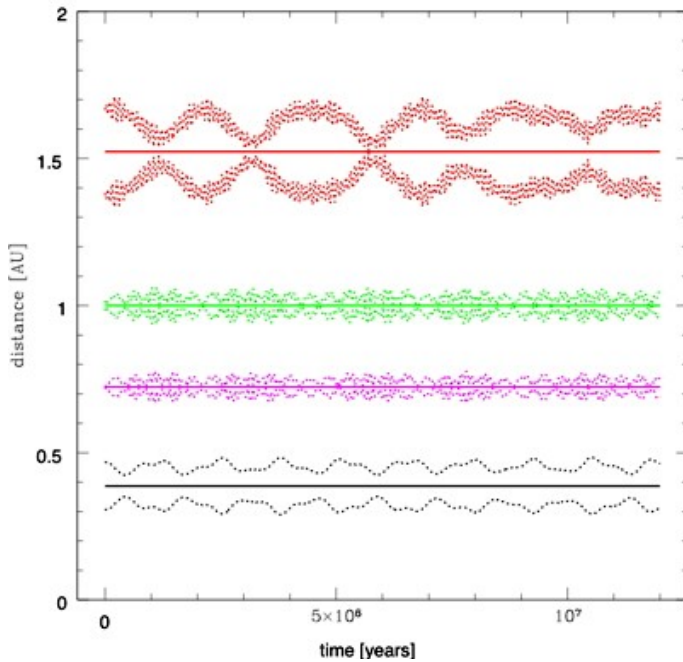
In grand scheme of things, though, you might think that nothing would change. According to Kepler's laws, the mass of a planet has almost no effect on its orbit; the mass of the sun is what controls things. Even though Earth is 10 times heavier than Mars, it would still trundle along Mars's old path. Both Mars and Earth are perpetually falling toward the sun, and all falling bodies fall at the same rate.

But Kepler's laws don't account for the subtle gravitational perturbations that planets exert on one another. By rearranging the planets, you perturb these perturbations, and it's not obvious what would happen. So I posed the question to planetary physicist Renu Malhotra of the University of Arizona, who was one of the first scientists to recognize that the planets migrated around early in the history of the solar system. Her initial guess was that Earth's proximity would thin out the asteroid belt, but that the planets' orbits would not be destabilized, at least not right away. She offered to run a computer simulation to check.

The results are a bit surprising. The planetary switch-a-roo makes the inner solar system strongly chaotic. Although none of the inner planets gets flung out of the solar system within the first 10 million years, all undergo large variations in their orbital distances. On occasion, Mars dips inward to become the second rock from the sun. To capture these variations, Malhotra found that she had to use a smaller time increment in the simulations than she had predicted, and consequently each computer run took nearly a day to complete.

To speed things up, she tried ignoring the planet Mercury—standard practice in perturbative calculations, on the assumption that Mercury is so piddling that its gravity is immaterial. Not in this case, though. Without Mercury, the other three inner planets went haywire in a few million years. Mars shot off into deep space. The sensitivity to Mercury's absence is further proof that the altered system would be strongly chaotic.

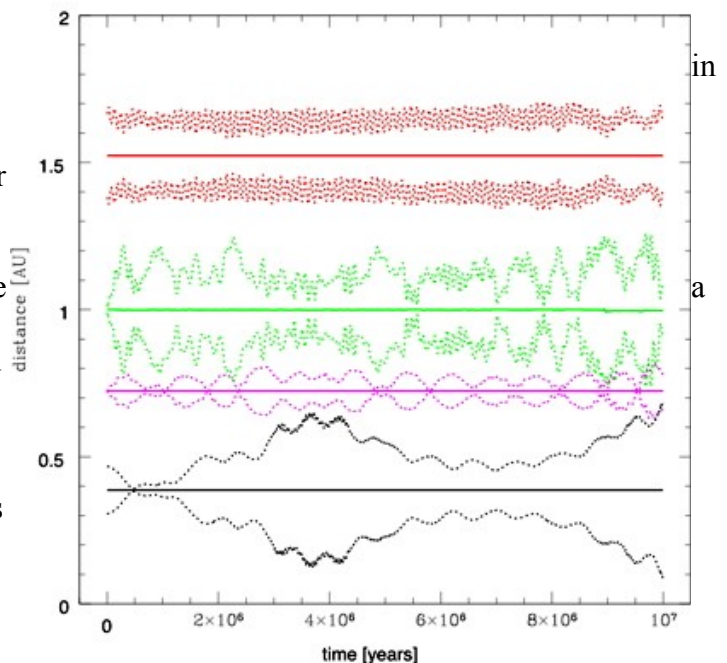
Solar System Stability
current solar system Mercury--Neptune



The graph at left shows the actual solar system. For each planet, Malhotra plots the range of orbital distances: perihelion (closest approach to the sun), aphelion (farthest) and semimajor axis (midpoint). As Pierre-Simon Laplace showed in the late 18th century, our solar system is stable. The semimajor axes are constant, and the shapes of orbits vary modestly on a variety of periods, from tens of thousands to millions of years.

The next graph shows the altered system. Notice how wide the range of orbital distances for each planet has become. For Earth, that's because it's now closer to Jupiter; for Mars, because it's the monkey in the middle. Venus changes hardly at all, while Mercury gets batted around like a pingpong ball. Malhotra's simulation also included the outer planets, but I leave them off, because they lumber on as if nothing had changed.

Alt Solar System Stability
Mercury--Neptune, Mars swapped with Earth



These results support the emerging view, discussed our pages by Doug Lin several years ago, that the solar system lives on the edge of chaos. It was probably unstable in its formative years. Planets got reshuffled or ejected until the survivors' orbits were sufficiently well spaced. Any major change would push the system over the edge again. It's analogous to a coffee cup. If you see cup that is filled exactly to the rim, you can reasonably conclude that some coffee got spilled over the side, and anything you do to the cup would probably spill some more.

Malhotra has supported this viewpoint in the past, but cautions that the solar system is more stable than its age might imply, so the whole question remains unresolved. "Isn't it interesting?" she wrote me. "This kind of thing is what attracted me to planetary dynamics."

<http://www.physorg.com/news/2011-06-survivors-joplin-tornado-rare-infection.html>

Survivors of Joplin tornado develop rare infection

(AP) -- In the aftermath of the Joplin tornado, some people injured in the storm developed a rare and sometimes fatal fungal infection so aggressive that it turned their tissue black and caused mold to grow inside their wounds.

Scientists say the unusually aggressive infection occurs when dirt or vegetation becomes embedded under the skin. In some cases, injuries that had been stitched up had to be reopened to clean out the contamination.

The Centers for Disease Control and Prevention said Friday that it was conducting tests to help investigate the infections, which are so uncommon that even the nation's largest hospitals might see only one or two cases a year. "To my knowledge, a cluster like this has not been reported before," said Dr. Benjamin Park, head of the CDC team that investigates fungal diseases. "This is a very rare fungus. And for people who do get the disease, it can be extremely severe."

Three tornado survivors who were hospitalized with the infection have died, but authorities said it was unclear what role the fungus played in their deaths because they suffered from a host of other serious ailments.

"These people had multiple traumas, pneumonia, all kinds of problems," said Dr. Uwe Schmidt, an infectious disease specialist at Freeman Health System in Joplin. "It's difficult to say how much the fungal infections contributed to their demise."

The infection develops in two ways: when the fungal spores are inhaled or when a tree branch or other object carrying the fungus pierces the flesh.

Most people who get sick by inhaling the spores already have weakened immune systems or diabetes. But healthy people can become sick if the fungus penetrates their skin. The fungus blocks off blood vessels to the infected area, causing tissue to turn red and begin oozing. Eventually it becomes black.

If diagnosed in time, the infection can be treated with intravenous medications and surgical removal of affected tissue. But it's considered exceptionally dangerous, with some researchers reporting fatality rates of 30 percent for people infected through wounds and 50 percent for susceptible people who breathe it in.

Small numbers of cases have been reported after some disasters, but Park said it's the particular circumstance of the wound - not the disaster itself - that creates the risk.

The Missouri Department of Health and Senior Services has received reports of eight suspected deep-skin fungal infections among survivors of the May 22 twister, which was the nation's deadliest single tornado in more than six decades. All of the patients had suffered multiple injuries. Also Friday, Joplin officials raised the death toll from the twister to 151, a figure that includes the recent deaths of the three people who had the fungus.

Schmidt said his hospital treated five Joplin tornado victims for the infection, which is formally known as zygomycosis (zy'-goh-my-KOH'-sihs). In 30 years of medical practice, he said, he had seen only two cases. Both involved patients with untreated diabetes.

Joplin officials say more than 1,100 people have been treated for injuries after the storm, many of them from objects sent flying by the twister. "These were very extensive wounds," Schmidt said. "They were treated in the emergency room as quickly as possible." A week after the tornado, patients began arriving with fungal infections. Doctors had to reopen some wounds that had been stitched closed because the injuries had not been adequately cleaned, Schmidt said.

After the infections set in, doctors "could visibly see mold in the wounds," Schmidt said. "It rapidly spread. The tissue dies off and becomes black. It doesn't have any circulation. It has to be removed."

The fungus "invades the underlying tissue and actually invades the underlying blood vessels and cuts off the circulation to the skin," he said. "It's very invasive."