http://www.eurekalert.org/pub_releases/2012-07/uoca-lsa072512.php

Later Stone Age got earlier start in South Africa than thought

Study led by CU-Boulder pushes back onset date of South Africa's Later Stone Age by more than 20,000 years

The Later Stone Age emerged in South Africa more than 20,000 years earlier than previously believed -- about the same time humans were migrating from Africa to the European continent, says a new international study led by the University of Colorado Boulder. The study shows the onset of the Later Stone Age in South Africa likely began some 44,000 to 42,000 years ago, said Paola Villa, a curator at the University of Colorado Museum of Natural History and lead study author. The new dates are based on the use of precisely calibrated radiocarbon dates linked to organic artifacts found at Border Cave in the Lebombo Mountains on the border of South Africa and Swaziland containing evidence of hominid occupation going back 200,000 years.

The Later Stone Age is synonymous to many archaeologists with the Upper Paleolithic Period, when modern humans moved from Africa into Europe roughly 45,000 years ago and spread rapidly, displacing and eventually driving Neanderthals to extinction. The timing of the technological innovations and changes in the Later Stone Age in South Africa are comparable to that of the Upper Paleolithic, said Villa.

"Our research proves that the Later Stone Age emerged in South Africa far earlier than has been believed and occurred at about the same time as the arrival of modern humans in Europe," said Villa. "But differences in technology and culture between the two areas are very strong, showing the people of the two regions chose very different paths to the evolution of technology and society."

A paper on the subject was published July 30 in the Proceedings of the National Academy of Sciences. Co-authors included Sylvain Soriano of the Center National de la Recherche Scientifique, or CNRS, at the University of Paris; Tsenka Tsanova of the Max Planck Institute of Evolutionary Biology in Leipzig, Germany; Ilaria Degano, Jeannette Lucejko and Maria Perla Colombini of the University of Pisa in Italy; Roger Higham of the University of Oxford in England; Francesco d'Errico of the CNRS at the University of Bordeaux in France; Lucinda Blackwell of the University of Witwatersrand in South Africa; and Peter Beaumont of the McGregor Museum in South Africa.

A companion paper published in PNAS and led by d'Errico reports on organic materials found at Border Cave dating to the Later Stone Age, an indication that the San hunter-gatherer culture first thought to have begun about 20,000 years ago in the region probably emerged as early as 44,000 years ago, said Villa.

Organic artifact assemblages at Border Cave dating to the Later Stone Age included ostrich eggshell beads, thin bone arrowhead points, wooden digging sticks, a gummy substance called pitch that was used to haft, or attach, bone and stone blades to shafts and a lump of beeswax likely used for hafting. The assemblage also included worked tusks of members of the pig family, which likely were used to plane wood, and notched bones that may have been used for counting.

A wooden digging stick from Border Cave dated to about 40,000 years ago was found in association with bored but broken stones likely used to weight such sticks. The sticks and stone weights are similar to digging implements used by women of the prehistoric San hunter-gatherer culture in the region to unearth bulbs and termite larvae, a practice that continued into historic times, said Villa. "These digging sticks from Border Cave are the oldest artifacts of this kind known from South Africa or anywhere else in Africa."

The new PNAS study led by Villa also indicates big changes were occurring in hunting technology during the Later Stone Age at Border Cave, said Villa. They included a shift from spears hafted with stone points -- the main hunting weapon in the Middle Stone Age -- to the likely use of the bow and arrow, a technology that included very thin bone points that probably were tipped with poison, she said.

"The very thin bone points from the Later Stone Age at Border Cave are good evidence for bow and arrow use," said Villa. "The work by d'Errico and colleagues shows that the points are very similar in width and thickness to the bone points produced by San culture that occupied the region in prehistoric times, whose people were known to use bows and arrows with poison-tipped bone points as a way to bring down medium and large-sized herbivores."

Chemical analyses showed the poison used with such bone points was most likely ricinoleic acid, which can be derived from the seeds of castor oil plants and which has been identified as being used in South Africa at least 24,000 years ago. "Such bone points could have penetrated thick hides, but the lack of 'knock-down' power means the use of poison probably was a requirement for successful kills," said Villa.

The lump of beeswax from Border Cave also dating to about 40,000 years ago -- the oldest known beeswax used by humans ever discovered -- was wrapped in plant fibers that may have been similar to fibers used to make the strings for hunting bows, said Villa.

While stone tools continued to be manufactured in the Later Stone Age at Border Cave, stone spear points from the Middle Stone Age gave way to tiny, thin flakes known as microliths that were probably hafted on shafts, much like the bone points, with pitch made from the bark of a common type of coniferous tree found in the region.

While a 2011 study co-authored by Villa and Wil Roebroeks of Leiden University in the Netherlands showed that Neanderthals mastered the manufacture of pitch in Europe 200,000 years ago, it was not a particularly simple task since the process involved burning peeled bark in the absence of air, said Villa. The Later Stone Age inhabitants of South Africa probably dug holes into the ground and inserted bark peels, then lit them on fire and covered the holes tightly with stones. "This is the first time pitch-making is demonstrated in South Africa," said Villa.

The Upper Paleolithic Period in Europe that corresponds to the Later Stone Age in South Africa also spurred complex new technologies that helped humans survive and thrive in much different environments. Artifacts from the Upper Paleolithic included spear-throwers, bone needles with eyelets for sewing furs, bone fishing hooks, bone flutes and even ivory figurines carved from mammoth tusks. Villa said that a fundamental rearrangement of human behavior that had its beginnings 50,000-60,000 years ago in Africa and spread to Europe -- an idea first proposed by Stanford University archaeologist Richard Klein -- appears quite plausible.

Research at Border Cave was funded by the National Science Foundation, the Paleontological National Trust in South Africa, the School of Geography, Archaeology and Environment at the University of Witwatersrand in South Africa and the French National Centre for Scientific Research Laboratory at the University of Bordeaux.

http://www.eurekalert.org/pub_releases/2012-07/uoc--bad073012.php

Brains are different in people with highly superior autobiographical memory

UCI study finds structural variations among those who recall their lives perfectly

Irvine, Calif – UC Irvine scientists have discovered intriguing differences in the brains and mental processes of an extraordinary group of people who can effortlessly recall every moment of their lives since about age 10.

The phenomenon of highly superior autobiographical memory – first documented in 2006 by UCI neurobiologist James McGaugh and colleagues in a woman identified as "AJ" – has been profiled on CBS's "60 Minutes" and in hundreds of other media outlets. But a new paper in the peer-reviewed journal Neurobiology of Learning & Memory's July issue offers the first scientific findings about nearly a dozen people with this uncanny ability.

All had variations in nine structures of their brains compared to those of control subjects, including more robust white matter linking the middle and front parts. Most of the differences were in areas known to be linked to autobiographical memory, "so we're getting a descriptive, coherent story of what's going on," said lead author Aurora LePort, a doctoral candidate at UCI's Center for the Neurobiology of Learning & Memory.

Surprisingly, the people with stellar autobiographical memory did not score higher on routine laboratory memory tests or when asked to use rote memory aids. Yet when it came to public or private events that occurred after age 10½, "they were remarkably better at recalling the details of their lives," said McGaugh, senior author on the new work.

"These are not memory experts across the board. They're 180 degrees different from the usual memory champions who can memorize pi to a large degree or other long strings of numbers," LePort noted. "It makes the project that much more interesting; it really shows we are homing in on a specific form of memory."

She said interviewing the subjects was "baffling. You give them a date, and their response is immediate. The day of the week just comes out of their minds; they don't even think about it. They can do this for so many dates, and they're 99 percent accurate. It never gets old."

The study also found statistically significant evidence of obsessive-compulsive tendencies among the group, but the authors do not yet know if or how this aids recollection. Many of the individuals have large, minutely catalogued collections of some sort, such as magazines, videos, shoes, stamps or postcards.

UCI researchers and staff have assessed more than 500 people who thought they might possess highly superior autobiographical memory and have confirmed 33 to date, including the 11 in the paper. Another 37 are strong candidates who will be further tested.

"The next step is that we want to understand the mechanisms behind the memory," LePort said. "Is it just the brain and the way its different structures are communicating? Maybe it's genetic; maybe it's molecular."

McGaugh added: "We're Sherlock Holmeses here. We're searching for clues in a very new area of research."

Fellow authors are Aaron Mattfeld, Heather Dickinson-Anson, James Fallon, Craig Stark, Frithjof Kruggel and Larry Cahill. Funding was provided by the National Institutes of Health, the Gerard Family Trust and Unither Neurosciences Inc.

http://www.scientificamerican.com/article.cfm?id=can-bacteria-fight-brain-cancer

Can Bacteria Fight Brain Cancer?

The thinking behind an approach that has caused trouble in California

By Monya Baker of Nature magazine

Last week, the Sacramento Bee reported that two neurosurgeons at the University of California, Davis, had been banned from research on humans after deliberately infecting three terminally ill cancer patients with pathogenic bacteria in an attempt to treat them. All three died, two showing complications from the infection. Nature explores what happened and the science behind it.

Who authorized the researchers to infect the patients?

All three patients consented to infection. However, anyone testing experimental drugs in the United States requires approval from their university's Institutional Review Board (IRB) and oversight by the country's Food and Drug Administration (FDA), both of which review evidence for safety and efficacy. Neurosurgeons Paul Muizelaar and Rudolph Schrot at the University of California (UC), Davis, did not obtain this approval; they say they did not think it was required. Harris Lewin, the vice-chancellor of research at UC Davis, wrote a letter to the FDA describing what had occurred as "serious and continuing noncompliance".

In 2008, working under instructions from Muizelaar, Schrot asked the FDA about the possibility of deliberately infecting a postoperative wound in a particular patient with glioblastoma with the bacterium Enterobacter aerogenes. He was told that animal studies were needed first. Muizelaar did not infect that patient, but arranged for a graduate student to begin tests in rats. Although bacteria were purchased as research materials not to be used in humans, they were eventually used in three other patients with glioblastoma.

The first of those asked Muizelaar about infection in 2010, and Schrot contacted the director of UC Davis's IRB asking permission to perform what Lewin's letter describes as a "one-time procedure" not intended as research. The director concluded that this procedure could be classed as "innovative care" that did not require approval by the FDA or IRB, but that subsequent work should be reviewed. Schrot and Muizelaar went on to treat two further patients and were seeking approval from an ad hoc ethics committee (not the IRB or FDA) to treat five more when the IRB director told the neurosurgeons to cease and desist, and began an internal investigation.

How is brain cancer usually treated?

Glioblastoma is an aggressive brain cancer and is usually treated with surgery, as well as with radiation and chemotherapy. However, the cancer almost always recurs after surgery. Half of patients die within 15 months of diagnosis; fewer than one in twenty lives longer than five years.

Why might an infection fight cancer?

There are isolated reports of patients with various types of cancer successfully fighting off an infection only to find that the cancer has also disappeared. Presumably, the infection spurs white blood cells to attack both pathogens and malignant cells. In 1999, researchers at the University of Mississippi Medical Center in Jackson described four case studies in which the regression of malignant brain tumors co-occurred with infection. Enterobacter aerogenes, the same bacterium used at UC Davis, was recovered from microbial cultures taken from three of the patients.

What further studies have been done?

In 2004, a group led by Bert Vogelstein at the Howard Hughes Medical Institute in Maryland introduced cancer cells into mice and rabbits, allowed large tumors to form and then injected the animals with spores of the anaerobic bacterium Clostridium novyi-NT. About one-third of the animals' tumors disappeared, apparently as a result of an immune response.

Then, in 2011, researchers at the Catholic University of Rome examined the records of 197 patients treated for glioblastoma between 2001 and 2008, of which ten developed pathogenic infections after surgery. Those patients had a median survival rate of 30 months, whereas patients who did not become infected had a median survival rate of 16 months. However, the authors concluded that the association was "not definitive".

A 2009 report considered 382 patients with malignant brain cancer, 18 of whom developed infections. Infected patients lived longer on average, but the difference was not statistically significant. What's more, the researchers reasoned that infection may correlate with longer survival not because infection prolongs survival but because patients who live longer are more likely to develop infections.

Are there clinical trials studying whether an immune response can fight cancer?

Yes. But rather than infecting patients with active microbes, these studies use therapeutic vaccines. The first cancer-treatment vaccine, Provenge, for prostate cancer, was approved in 2010 but is still controversial.

A US government registry of clinical trials that have attained regulatory approval lists more than three dozen studies using vaccines for glioblastoma. One, at Duke University in Durham, North Carolina, injects patients' brains with weakened, engineered poliovirus. Many of the others work by collecting a patient's white blood cells and exposing them to cancer-specific molecules.

http://www.sciencedaily.com/releases/2012/07/120730142509.htm

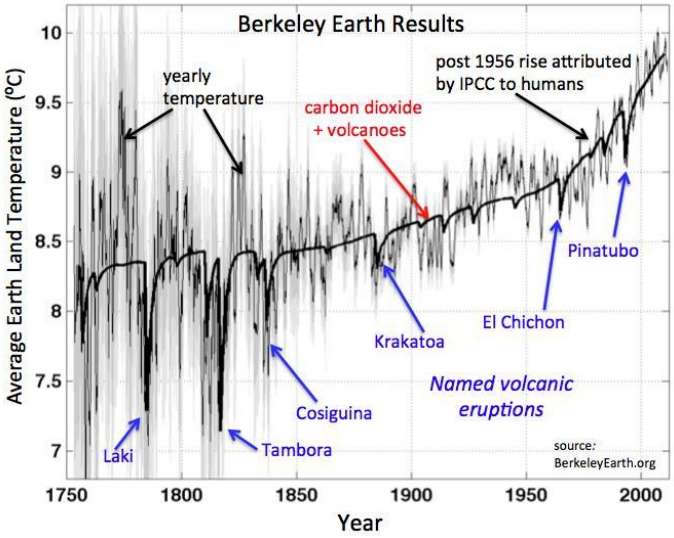
250 Years of Global Warming: Berkeley Earth Releases New Analysis

According to a new Berkeley Earth study released July 29, 2012, the average temperature of Earth's land has risen by 1.5 °C over the past 250 years.

ScienceDaily - The good match between the new temperature record and historical carbon dioxide records suggests that the most straightforward explanation for this warming is human greenhouse gas emissions.

Together with their most recent results and papers, Berkeley Earth also released their raw data and analysis programs. They will be available online at BerkeleyEarth.org on July 30.

The new analysis from Berkeley Earth goes all the way back to 1753, about 100 years earlier than previous groups' analyses. The limited land coverage prior to 1850 results in larger uncertainties in the behavior of the record; despite these, the behavior is significant.



The temperature of the Earth's land surface, as determined from over 36,000 temperature stations around the globe. The data is well fit by a simple model containing only known volcanic eruptions and carbon dioxide (dark line). No contribution from solar variability was necessary to make a good match. The rapid but short (decadal) variations are believed to be due to changes in ocean flows, such as El Nino and the Gulf Stream. (Credit: Image courtesy of Berkeley Earth Surface Temperature)

Robert Rohde, Lead Scientist for Berkeley Earth and the person who carried out most of the analysis, noted that "Sudden drops in the early temperature record (1753 to 1850) correspond to known volcanic events."

Volcanoes spew particles into the air, which then reflect sunlight and cool the earth for a few years. In the Berkeley Earth temperature plot, sudden dips in temperature caused by large volcanic explosions are evident back to the late 1700s.

Berkeley Earth compared the shape of the gradual rise over 250 years to simple math functions (exponentials, polynomials) and to solar activity (known through historical records of sunspot numbers), and even to rising functions such as world population.

Richard Muller, Founder and Scientific Director of Berkeley Earth, notes "Much to my surprise, by far the best match was to the record of atmospheric carbon dioxide, measured from atmospheric samples and air trapped in polar ice." He emphasizes that the match between the data and the theory doesn't prove that carbon dioxide is responsible for the warming, but the good fit makes it the strongest contender. "To be considered seriously, any alternative explanation must match the data at least as well as does carbon dioxide."

In its 2007 report the IPCC concluded only that "most" of the warming of the past 50 years could be attributed to humans. It was possible, according to the IPCC, that increased solar activity could have contributed to warming prior to 1956. Berkeley Earth analyzed about 5 times more station records than were used in previous analyses, and this expanded data base along with its new statistical approach allowed Berkeley Earth to go about 100 years farther back in time than previous studies. By doing so, the Berkeley Earth team was able to conclude that over 250 years, the contribution of solar activity to global warming is negligible.

Some of the scientists on the Berkeley Earth team admit surprise that the new analysis has shown such clear agreement between global land-− - temperature rise and human-− - caused greenhouse gases. "I was not expecting this," says Richard Muller, "but as a scientist, I feel it is my duty to let the evidence change my mind."

Elizabeth Muller, cofounder and Executive Director of Berkeley Earth, says that "One of our goals at Berkeley Earth is complete transparency - we believe that everyone should be able to access raw climate data and do their own analysis. Scientists have a duty to be 'properly skeptical', and we are trying to lower the barriers to entry into the field."

Robert Rohde created an online feature that allows look up temperature records by location. "If you want to know what the temperature change has been in your city, your state, or even your country, you can now find this online at BerkeleyEarth.org" says Rohde. He adds, "We hope people will have a lot of fun interacting with the data." This feature should be available to the public by Monday, July 30.

A previous Berkeley Earth study, released in October 2011, found that the land-− ‐ surface temperature had risen by about 0.9 ° C over the past 50 years (which was consistent with previous analyses) and directly addressed scientific concerns raised by skeptics, including the urban heat island effect, poor station quality, and the risk of data selection bias.

The Berkeley Earth team values the simplicity of its analysis, which does not depend on the large complex global climate models that have been criticized by climate skeptics for their hidden assumptions and adjustable parameters. The conclusion that the warming is due to humans is based simply on the close agreement between the shape of the observed temperature rise and the known greenhouse gas increase.

Elizabeth adds, "The current data does not include ocean temperatures; these will be added in the next phase of the Berkeley Earth studies. Another next step for our team is to think about the implications of our findings."

http://phys.org/news/2012-07-coating-percent-bacterial-slime-surfaces.html

New coating prevents more than 99 percent of harmful bacterial slime from forming on surfaces

Biofilms may no longer have any solid ground upon which to stand.

A team of Harvard scientists has developed a slick way to prevent the troublesome bacterial communities from ever forming on a surface. Biofilms stick to just about everything, from copper pipes to steel ship hulls to glass catheters. The slimy coatings are more than just a nuisance, resulting in decreased energy efficiency, contamination of water and food supplies, and - especially in medical settings - persistent infections. Even cavities in teeth are the unwelcome result of bacterial colonies.

In a study published in the Proceedings of the National Academy of Sciences (PNAS), lead coauthors Joanna Aizenberg, Alexander Epstein, and Tak-Sing Wong coated solid surfaces with an immobilized liquid film to trick the bacteria into thinking they had nowhere to attach and grow.

"People have tried all sorts of things to deter biofilm build-up - textured surfaces, chemical coatings, and antibiotics, for example," says Aizenberg, Amy Smith Berylson Professor of Materials Science at the Harvard School of Engineering and Applied Sciences (SEAS) and a Core Faculty Member at the Wyss Institute for Biologically Inspired Engineering at Harvard. "In all those cases, the solutions are short-lived at best. The surface treatments wear off, become covered with dirt, or the bacteria even deposit their own coatings on top of the coating intended to prevent them. In the end, bacteria manage to settle and grow on just about any solid surface we can come up with."

The word "SLIPS" is coated with the SLIPS technology to show its ability to repel liquids and solids and even prevent ice or frost from forming. The slippery discovery has now been shown to prevent more than 99 percent of harmful bacterial slime from forming on surfaces. Credit: Joanna Aizenberg, Rebecca Belisle, and Tak-Sing Wong

Taking a completely different approach, the researchers used their recently developed technology, dubbed SLIPS (Slippery-Liquid-Infused Porous Surfaces) to effectively create a hybrid surface that is smooth and slippery due to the liquid layer that is immobilized on it.

First described in the September 22, 2011, issue of the journal Nature, the super-slippery surfaces have been shown to repel both water- and oil-based liquids and even prevent ice or frost from forming.

"By creating a liquid-infused structured surface, we deprive bacteria of the static interface they need to get a grip and grow together into biofilms," says Epstein, a recent Ph.D. graduate who worked in Aizenberg's lab at the time of the study.

"In essence, we turned a once bacteria-friendly solid surface into a liquid one. As a result, biofilms cannot cling to the material, and even if they do form, they easily 'slip' off under mild flow conditions," adds Wong, a researcher at SEAS and a Croucher Foundation Postdoctoral Fellow at the Wyss Institute.

Aizenberg and her collaborators reported that SLIPS reduced by 96% the formation of three of the most notorious, disease-causing biofilms - Pseudomonas aeruginosa, Escherichia coli, and Staphylococcus aureus - over a 7-day period.

The technology works in both a static environment and under flow, or natural conditions, making it ideally suited for coating implanted medical devices that interact with bodily fluids. The coated surfaces can also combat bacterial growth in environments with extreme pH levels, intense ultraviolet light, and high salinity.

SLIPS is also nontoxic, readily scalable, and - most importantly - self-cleaning, needing nothing more than gravity or a gentle flow of liquid to stay unsoiled. As previously demonstrated with a wide variety of liquids and solids, including blood, oil, and ice, everything seems to slip off surfaces treated with the technology.
To date, this may be the first successful test of a nontoxic synthetic surface that can almost completely prevent the formation of biofilms over an extended period of time. The approach may find application in medical, industrial, and consumer products and settings.
In future studies, the researchers aim to better understand the mechanisms involved in preventing biofilms. In particular, they are interested in whether any bacteria transiently attach to the interface and then slip off, if they just float above the surface, or if any individuals can remain loosely attached.
"Biofilms have been amazing at outsmarting us. And even when we can attack them, we often make the situation worse with toxins or chemicals. With some very cool, nature-inspired design tricks we are excited about the possibility that biofilms may have finally met their match," concludes Aizenberg.
Provided by Harvard University

http://phys.org/news/2012-07-humans-hard-wired-thy-neighbor.html

Humans might be hard-wired to 'love thy neighbor'

Researchers report people were less likely to punish those standing closer to them.

HealthDay - The amount of physical space between people may influence how they react to each other in certain situations, new research suggests. British psychologists from the University of Lincoln argue that people may actually be hard-wired to "love thy neighbor." In conducting the study, the researchers analyzed the behavior of contestants in first-round episodes of the BBC quiz show, "The Weakest Link."
"In the show contestants must make a choice about who is the worst player based on two very different sources of information," study leader Paul Goddard, senior lecturer in the School of Psychology, explained in a Lincoln news release. "The primary and most reliable source comes from the game itself. If one player gets all their questions wrong, it's a fairly straightforward decision to vote them off. The quandary for contestants arises when there is no clear consensus about who is the worst player, such as in rounds where several players get just one question wrong. In these circumstances, contestants have to rely on a secondary source of information -- their own judgment. This is where bias can really come to the fore."
The researchers calculated the probability of votes and compared these projections to what actually happened. The study found contestants showed a strong reluctance to vote for the person standing next to them. The researchers dubbed this pattern, 'the neighbor avoidance effect.' They noted this bias was stronger when the group of contestants didn't agree on which players was the weakest.
When forced to make decisions, the study revealed people were less likely to vote off the people next to them and target other contestants who were standing farther away.
The researchers said their observations drew parallels from a controversial social psychology experiment conducted in the 1960s. In this experiment, Yale psychologist Stanley Milgram found people were more likely to punish people with an electric shock if they were in another room. If people were located in the same room however, they were more reluctant to administer this punishment.
Aside from the distance between players, the researchers found evidence of a gender bias in voting patterns as well. Men and women, they found, were more likely to vote off a woman than a man.
The study was presented recently at the 2012 Society for the Advancement of Behavioral Economics Conference in Granada, Spain. Data and conclusions should be viewed as preliminary until published in a peer-reviewed journal.

http://www.eurekalert.org/pub_releases/2012-07/jaaj-sfc072612.php

Study finds correlation between number of colorectal polyps and genetic mutations

Among patients with multiple colorectal polyps, the prevalence of certain gene mutations varied considerably by polyp count

CHICAGO – Among patients with multiple colorectal polyps, the prevalence of certain gene mutations varied considerably by polyp count, according to a study in the August 1 issue of JAMA. "Patients with multiple colorectal adenomas [polyps] may carry germline [those cells of an individual that have genetic material that could be passed to offspring] mutations in the APC or MUTYH genes," according to background information in the article. The authors write that guidelines for when genetic evaluation should be performed in individuals with multiple colorectal adenomas vary, and data to support such guidelines are limited.
Shilpa Grover, M.D., M.P.H., of Brigham and Women's Hospital, Boston, and colleagues conducted a study to evaluate the frequency of APC and MUTYH mutations by the number of colorectal adenomas among individuals who had undergone clinical genetic testing. The researchers also studied the relationship between

the number of adenomas and age at diagnosis of adenoma and colorectal cancer and the prevalence of pathogenic APC or MUTYH mutations. The study included 8,676 individuals who had undergone full gene sequencing between 2004 and 2011. Individuals with a certain mutation of the MUTYH gene (Y179C and G396D) underwent full MUTYH gene sequencing. APC and MUTYH mutation prevalence was evaluated by the number of polyps.

Colorectal adenomas were reported in 7,225 individuals; 1,457 with classic polyposis (100 adenomas or more) and 3,253 with attenuated (diminished) polyposis (20-99 adenomas). "The prevalence of pathogenic APC and biallelic [pertaining to both alleles (both alternative forms of a gene)] MUTYH mutations was 95 of 119 (80 percent) and 2 of 119 (2 percent), respectively, among individuals with 1,000 or more adenomas, 756 of 1,338 (56 percent) and 94 of 1,338 (7 percent) among those with 100 to 999 adenomas, 326 of 3,253 (10 percent) and 233 of 3,253 (7 percent) among those with 20 to 99 adenomas, and 50 of 970 (5 percent) and 37 of 970 (4 percent) among those with 10 to 19 adenomas. Adenoma count was strongly associated with a pathogenic mutation in multivariable analyses," the authors write.

The researchers note that their evaluation of individuals who underwent genetic testing because of a personal or family history suggestive of a familial polyposis syndrome suggests that genetic evaluation for APC and MUTYH mutations may be considered in individuals with 10 or more adenomas. "However, our results are derived from a selected cohort of high-risk individuals and need to be validated in larger populations of unselected patients."

"The mutation probabilities reported here may assist clinicians in their decision to recommend genetic evaluation and counsel patients undergoing genetic testing. However, it remains important to also consider the limitations of genetic testing at present, because one-third of patients with a classic familial adenomatous polyposis [FAP; a polyposis syndrome resulting from mutations in the APC gene characterized by multiple colorectal polyps] phenotype are found to not carry a mutation in either the APC or MUTYH gene. Such individuals should undergo periodic re-evaluation as other susceptibility genes are identified."

(JAMA. 2012;308[5]:485-492. Available pre-embargo to the media at http://media.jamanetwork.com)

Editor's Note: This study was supported by National Cancer Institute grants and by a National Institutes of Health grant. Please see the article for additional information, including other authors, author contributions and affiliations, financial disclosures, etc.

Editorial: APC Gene Testing for Familial Adenomatosis Polyposis

"At this juncture, clinicians need to carefully consider the effect of a positive or negative test result on management of patient care prior to making decisions regarding genetic testing," write Hemant K. Roy, M.D., and Janardan D. Khandekar, M.D., of the NorthShore University HealthSystem, Evanston, Il., in an accompanying editorial.

"Appropriate patient education and informed consent prior to testing is mandatory, highlighting the integral nature of genetic counseling. Until development of more robust genomic technologies for FAP detection, complementary approaches including careful assessment of family history and biomarkers may have utility. Furthermore, these considerations for FAP may serve as a model for evaluating the wider issues associated with practicing medicine at the front lines of the genomic revolution."

(JAMA. 2012;308[5]:514-515. Available pre-embargo to the media at http://media.jamanetwork.com)

http://www.eurekalert.org/pub_releases/2012-07/afps-wwf073112.php

When we forget to remember - failures in prospective memory range from annoying to lethal

Why would highly skilled professionals forget to perform a simple task they have executed without difficulty thousands of times before?

A surgical team closes an abdominal incision, successfully completing a difficult operation. Weeks later, the patient comes into the ER complaining of abdominal pain and an X-ray reveals that one of the forceps used in the operation was left inside the patient. Why would highly skilled professionals forget to perform a simple task they have executed without difficulty thousands of times before?

These kinds of oversights occur in professions as diverse as aviation and computer programming, but research from psychological science reveals that these lapses may not reflect carelessness or lack of skill but failures of prospective memory.

In an article in the August issue of Current Directions in Psychological Science, a journal of the Association for Psychological Science, R. Key Dismukes, a scientist at the NASA Ames Research Center, reviews the rapidly growing field of research on prospective memory, highlighting the various ways in which characteristics of everyday tasks interact with normal cognitive processes to produce memory failures that sometimes have disastrous consequences.

Failures of prospective memory typically occur when we form an intention to do something later, become engaged with various other tasks, and lose focus on the thing we originally intended to do. Despite the name, prospective memory actually depends on several cognitive processes, including planning, attention, and task management. Common in everyday life, these memory lapses are mostly annoying, but can have tragic consequences. "Every summer several infants die in hot cars when parents leave the car, forgetting the child is sleeping quietly in the back seat," Dismukes points out.

Many examples of prospective memory involve intending to do something at a particular time, such as going to a doctor's appointment, or on a particular occasion, such as congratulating a friend the next time you see her. However, much of what we intend to do in our everyday lives, whether at home or at work, involves habitual tasks repeated over time. And when it comes to these kinds of habitual tasks, our intentions may not be explicit. We usually don't, for example, form an explicit intention to insert the key in the ignition every time we drive a car - the intention is implicit in our habitual routine of driving.

In previous research, Dismukes and colleagues identified several types of situations that can lead to prospective memory failures. They found that interruptions and disruptions to habitual processes, which are irritating enough in everyday life, can be fatal in some occupational settings. In fact, several airline catastrophes have occurred because pilots were interrupted while performing critical preflight tasks – after the interruption was over, the pilots skipped to the next task, not realizing that the interrupted tasks hadn't been finished.

For all the negative attention that multitasking has received in recent years, it is perhaps no surprise that multitasking is also a major cause of prospective memory failures. We seem to have adapted fairly well to juggling several tasks simultaneously. But research shows that when a problem arises with whatever task we're currently focused on, we become vulnerable to cognitive tunneling, forgetting to switch our attention back to the other tasks at hand.

To defend against prospective memory failures and their potentially disastrous consequences, professionals in aviation and medicine now rely on specific memory tools, including checklists. Research also reveals that implementation intentions, identifying when and where a specific intention will be carried out, can help guard against such failures in everyday life. Dismukes points out that having this kind of concrete plan has been shown to improve prospective memory performance by as much as two to four times in tasks such as exercising, medication adherence, breast self-examination, and homework completion.

Along with checklists and implementation intentions, Dismukes and others have highlighted several other measures that can help to remember and carry out intended actions:

Use external memory aids such as the alerting calendar on cell phones
Avoid multitasking when one of your tasks is critical
Carry out crucial tasks now instead of putting them off until later
Create reminder cues that stand out and put them in a difficult-to-miss spot
Link the target task to a habit that you have already established

"Rather than blaming individuals for inadvertent lapses in prospective memory, organizations can improve safety by supporting the use of these measures," argues Dismukes. He suggests that scientists should combine laboratory research with observations of human performance in real-world settings to better understand how prospective memory works and to develop practical strategies to avoid lapses.

http://www.eurekalert.org/pub_releases/2012-07/foas-ays073112.php

Allergies? Your sneeze is a biological response to the nose's 'blue screen of death'

New research in the FASEB Journal suggests that sneezing is the body's natural reboot and that patients with disorders of the nose such as sinusitis can't reboot, explaining why they sneeze more often than others

Who would have thought that our noses and Microsoft Windows' infamous blue screen of death could have something in common? But that's the case being made by a new research report appearing online in The FASEB Journal (http://www.fasebj.org). Specifically, scientists now know exactly why we sneeze, what sneezing should accomplish, and what happens when sneezing does not work properly. Much like a temperamental computer, our noses require a "reboot" when overwhelmed, and this biological reboot is triggered by the pressure force of a sneeze. When a sneeze works properly, it resets the environment within nasal passages so "bad" particles breathed in through the nose can be trapped. The sneeze is accomplished by biochemical signals that regulate the beating of cilia (microscopic hairs) on the cells that line our nasal cavities. "While sinusitis rarely leads to death, it has a tremendous impact on quality of life, with the majority of symptoms coming from poor clearance of mucus," said Noam A. Cohen, M.D., Ph.D., a researcher involved in the work from the Department of Otorhinolaryngology-Head and Neck Surgery at the University of

Pennsylvania in Philadelphia. "By understanding the process by which patients with sinusitis do not clear mucus from their nose and sinuses, we can try to develop new strategies to compensate for their poor mucus clearance and improve their quality of life."
To make this discovery, Cohen and colleagues used cells from the noses of mice which were grown in incubators and measured how these cells cleared mucus. They examined how the cells responded to a simulated sneeze (puff of air) by analyzing the cells' biochemical responses. Some of the experiments were replicated in human sinus and nasal tissue removed from patients with and without sinusitis. They found that cells from patients with sinusitis do not respond to sneezes in the same manner as cells obtained from patients who do not have sinusitis. The researchers speculate that sinusitis patients sneeze more frequently because their sneezes fail to reset the nasal environment properly or are less efficient at doing so. Further understanding of why sinusitis patients have this difficulty could aid in the development of more effective medications or treatments.
"I'm confident that modern biochemical studies of ciliary beating frequency will help us find new treatments for chronic sinusitis," said Gerald Weissmann, M.D., Editor-in-Chief of The FASEB Journal, "I'm far less confident in our abilities to resolve messy computer crashes. We now know why we sneeze. Computer crashes are likely to be a mystery forever."

http://www.scientificamerican.com/article.cfm?id=high-trans-fat-diet-predicts-aggresion

High Trans-Fat Diet Predicts Aggression

People who eat more hydrogenated oils are more aggressive

By Winnie Yu | Tuesday, July 31, 2012 | 5

If you want to keep your cool, you might want to pass up those greasy wings and gooey dessert. A new study from the University of California, San Diego, suggests that people whose diets are higher in trans fats are more prone to aggression. Trans fats, or hydrogenated oils, have made the news in recent years because studies have strongly linked them to heart disease and cancer, and some locales have passed laws restricting their use. They are still common, however, in restaurant food and many grocery items.
Beatrice Golomb, a physician and associate professor of medicine at U.C. San Diego, wondered if trans fats might affect behavior, after noting how they interact with a type of healthy fat. Past studies found that docosahexaenoic acid - or DHA, a long-chain omega-3 fatty acid - has a calming, antidepressant effect. Trans fats disrupt the chemical process that leads to the conversion of fatty acids into DHA, which led Golomb to suspect that trans fats might be linked to aggression.
Her study, which was published in March in PLoS ONE, involved 1,018 men and women older than 20 who filled out a food questionnaire and several other surveys that measure impatience, irritability and aggression. Even after considering other influences, Golomb's team found a strong link between the intake of trans fats and aggression. “Trans-fatty acids were a more consistent predictor of aggression than some traditional risk factors such as age, male sex, education and smoking,” Golomb says. The findings were consistent across both sexes and across all ages, ethnicities and socioeconomic groups.
Although the correlation was strong, the study does not prove that trans fats are causing the aggressive behavior. It is possible that naturally aggressive people tend to eat less healthy food. Or perhaps other ingredients found in processed foods, such as added sugars, are the real culprit. “We like to think we're in charge of our behaviors, but in fact there are many factors that influence us, food being one of them,” Golomb says.

http://nyti.ms/RnCESd

Another Tick-Borne Disease to Guard Against

Babesiosis, has only recently been widely recognized as a potentially serious outdoor hazard

By JANE E. BRODY

Despite its many delights, summer also brings its fair share of pestilence. One, called babesiosis, has only recently been widely recognized as a potentially serious outdoor hazard. According to a very detailed study conducted on Block Island, R.I., it could eventually rival Lyme disease as the most common tick-borne ailment in the United States.But with reasonable precautions, neither babesiosis nor Lyme should keep you from enjoying a romp in the grass or hike in the woods.
Babesiosis is caused by protozoans that invade red blood cells and can cause a malarialike illness. The disease has an interesting history, recently recounted in The New England Journal of Medicine by Dr. Peter J. Krause, a Yale researcher specializing in tick-borne diseases, and Edouard Vannier, an immunologist at Tufts Medical Center.
Babesiosis (pronounced buh-BEEZ-e-OH-sis) is named for Dr. Victor Babes, a Romanian pathologist who in 1888 identified the disease in cattle that had fever and blood-tainted urine. Until the mid-20th century, the disease was known only in wild and domestic animals, which can be infected by more than 100 different

Babesia species. The first human case was not recognized until 1957. A Croatian herdsman who had no spleen, an important immunological organ, died quickly of the infection, which he most likely acquired from the animals he tended. Twelve years later, the first case in an immunologically normal person was identified on Nantucket Island, and for years the disease was called Nantucket fever.

Unlike Lyme disease, which quickly leapfrogged across the country, babesiosis is spreading slowly through the Northeast and Upper Midwest, where it is increasingly recognized as the cause of a flulike summer ailment. It has been said that Lyme disease moves on the wings of birds, which some experts believe carry the bacteria causing the condition. Babesiosis, however, moves on the backs of mice and deer. Birds do not spread it.

But like the bacteria that cause Lyme disease, Babesia protozoans are transmitted to humans by ticks, which acquire the infection from the white-footed mouse and white-tailed deer. And, yes, the same tick - Ixodes scapularis, popularly called a deer tick - transmits both Lyme disease and babesiosis in this country.

The deer tick, which starts out the size of a poppy seed, requires a blood meal at every one of its developmental stages. With its hind legs clutching grass or a leaf, the tick sits patiently, holding its pincerlike front legs extended, ready to latch onto an unsuspecting mammal that happens by.

In spring or summer, that mammal could be you. In the fall, the adult tick feeds on white-tailed deer, which don't get sick. This feeding allows female ticks to produce a profusion of eggs for the next generation.

Still Uncommon, but Worrisome

"Babesiosis is already a worldwide disease, though the United States has the most cases so far," Dr. Krause said in an interview. "Its geographical distribution is growing, and we think over time it will become increasingly important relative to Lyme disease." In 2011, the first year of national surveillance, only 1,000 cases of babesiosis were reported. But Dr. Krause's study on Block Island, which tracked the risk of infection among 70 percent of the people living there, revealed that babesiosis was one-third as common as Lyme among those who developed symptoms and nearly as common as Lyme in asymptomatic people.

Underreporting of babesiosis is expected to continue for a long time. People with only mild symptoms are unlikely to see a doctor, and without a telltale sign like the bull's-eye rash of Lyme or a simple blood test for the infection, most doctors are unlikely to diagnose babesiosis correctly, Dr. Krause said.

One-quarter of infected adults and half of infected children were free of symptoms, his study found. Yet if they donated blood, they could transmit the infection to others, with potentially dire consequences. There is no widely used test to screen blood donors for infection with Babesia.

Likewise, an infected woman could transmit the protozoans to her child during pregnancy or delivery. The risk of a severe and possibly fatal infection is highest in newborns, adults over 50 and anyone with compromised immunity, including people with cancer, H.I.V. or a transplanted organ, and those missing the spleen.

Dr. Krause explained that the spleen "helps to clear organisms in blood that shouldn't be there." "It produces antibodies that attack the protozoans, which are then gobbled up by macrophages, and it acts like a sieve, screening out Babesia-infected blood cells, which are too big to get through and back into circulation," he said.

Prevention and Treatment

As with Lyme disease, precautions to prevent the bite of a Babesia-bearing tick include staying on cleared trails to minimize contact with leaf litter, brush and tall grass; wearing socks with long pants tucked into them and long-sleeved shirts (not the most pleasant approach on a steamy summer day); and applying repellent to exposed skin and clothing. Products containing DEET can be applied directly to the skin and sprayed on clothing; those containing pyrethrins should be used only on clothing and shoes.

Daily tick checks should be as routine as brushing teeth for people in environments that could harbor ticks. Check everywhere, using mirrors if necessary, including underarms, groin, navel, back of the neck, behind knees, between toes, behind and in ears, and on the scalp.

If a tick is found, without delay use fine-pointed tweezers to grasp it close to the skin and pull upward with steady, even pressure. Do not twist or yank it. Then clean the area and your hands with rubbing alcohol or soap and water.

People who are infected can become ill one to four weeks after a tick bite. Common symptoms include fever, malaise, fatigue, chills and sweats, headache, muscle and joint pain, loss of appetite, cough and nausea. A blood test may reveal anemia. Certain diagnosis comes from detecting the protozoan in a blood smear. Dr. Krause suggested that labs examine 300 microscopic fields before ruling out the disease. While the infection clears in some people without treatment, most require a combination of antibiotics, usually atovaquone (Mepron) and azithromycin (Zithromax), for 7 to 10 days. Dr. Krause said even patients with mild symptoms should be treated because they may become severely ill at a later time or spread the infection to others through donated blood.

This post has been revised to reflect the following correction:
Correction: July 30, 2012 An earlier version of this article misstated, using information from The New England Journal of Medicine, the nationality of Dr. Victor Babes. He was Romanian, not Hungarian.

http://www.sciencenews.org/view/generic/id/342711/title/DNA_hints_at_African_cousin_to_humans

DNA hints at African cousin to humans

Gene profiles suggest people interbred with a now-extinct species on the continent not that long ago

By Tina Hesman Saey

Expeditions to Africa may have brought back evidence of a hitherto unknown branch in the human family tree. But this time the evidence wasn't unearthed by digging in the dirt. It was found in the DNA of hunter-gatherer people living in Cameroon and Tanzania.

Buried in the genetic blueprints of 15 people, researchers found the genetic signature of a sister species that branched off the human family tree at about the same time that Neandertals did. This lineage probably remained isolated from the one that produced modern humans for a long time, but its DNA jumped into the Homo sapiens gene pool through interbreeding with modern humans during the same era that other modern humans and Neandertals were mixing in the Middle East, researchers report in the August 3 Cell.

The evidence for ancient interbreeding is surprisingly convincing, says Richard "Ed" Green, a genome biologist at the University of California, Santa Cruz. "There is a signal that demands explanation, and archaic admixture seems to be the most reasonable one at this point," he says.

Scientists have discovered that some people with ancestry outside Africa have DNA inherited from Neandertals or Denisovans, a mysterious group known only through DNA derived from a fossil finger bone found in a Siberian cave (SN: 6/5/10, p. 5; SN: 1/15/11, p.10).

But those researchers had DNA from fossils to guide their research. This time, researchers led by Sarah Tishkoff at the University of Pennsylvania in Philadelphia didn't have fossil DNA, or even fossils.

Tishkoff's group took DNA donated by 15 African hunter-gatherers - five Pygmies from Cameroon and five Hadza and five Sandawe from Tanzania - and compiled complete genetic blueprints for each person.

Population geneticist Joshua Akey of the University of Washington and his colleagues helped analyze the data. Using a statistical analysis, the team determined that about 2 percent of the DNA from the hunter-gatherers came from an unknown species of hominid that split from modern human ancestors about 1.1 million years ago. These long-lost human cousins must have then interbred with modern humans sometime before the common ancestral lineage of the three hunter-gatherer groups separated about 30,000 to 70,000 years ago, Akey says.

A separate study posted online July 23 on arXiv.org examined patterns of single DNA unit changes, known as SNPs, in 22 African groups. That study, by Joseph Pickrell of Harvard Medical School and colleagues, also presents evidence that some African groups, including the Hadza, may harbor DNA from unknown extinct hominids.

Other researchers aren't convinced that the DNA remnants identified are the genetic remains of a new species of human cousin. The DNA could have come from a genetically distinct group of modern humans that has since died out due to changes in their environment, diseases or confrontations with rival groups of humans, says Jean-Jacques Hublin, a paleoanthropologist at the Max Planck Institute for Evolutionary Anthropology in Leipzig, Germany.

Relatively recent interbreeding isn't the only explanation for the presence of this newly discovered DNA, says anthropological geneticist Paul Verdu of Stanford University. He thinks the DNA may be the genetic stamp left by a common ancestor of modern humans and another species. The DNA may have morphed so much in non-African groups, just by chance, that it is now unrecognizable.

http://www.eurekalert.org/pub_releases/2012-08/cwru-cso073112.php

CWRU School of Medicine researchers discover gene that permanently stops cancer cell proliferation

Researchers have discovered a mutant form of a gene that permanently stopped their proliferation and caused cell death without chemotherapeutic drugs

Researchers at Case Western Reserve University School of Medicine have discovered a mutant form of the gene, Chk1, that when expressed in cancer cells, permanently stopped their proliferation and caused cell death without the addition of any chemotherapeutic drugs. This study illustrates an unprecedented finding, that artificially activating Chk1 alone is sufficient to kill cancer cells.

"We have identified a new direction for cancer therapy and the new direction is leading us to a reduction in toxicity in cancer therapy, compared with chemotherapy or radiation therapy," said Dr. Zhang, assistant

professor, Department of Pharmacology at the School of Medicine, and member of the university's Case Comprehensive Cancer Center. "With this discovery, scientists could stop the proliferation of cancer cells, allowing physicians time to fix cells and genetic errors."

While studying the basic mechanisms for genome integrity, Dr. Zhang's team unexpectedly discovered an active mutant form of human Chk1, which is also a non-natural form of this gene. This mutation changed the protein conformation of Chk1 from the inactive form into an active form. Remarkably, the research team discovered that when expressed in cancer cells, this active mutant form of Chk1 permanently stopped cancer cell proliferation and caused cell death in petri dishes even without the addition of any chemotherapeutic drugs. The biggest advantage of this potential strategy is that no toxic chemotherapeutic drug is needed to achieve the same cancer killing effect used with a combination of Chk1 inhibitors and chemotherapeutic drugs.

Cells respond to DNA damage by activating networks of signaling pathways, termed cell cycle checkpoints. Central to these genome pathways is the protein kinase, called Chk1. Chk1 facilitates cell survival, including cancer cells, under stressful conditions, such as those induced by chemotherapeutic agents, by placing a temporary stop on the cell cycle progression and coordinating repair programs to fix the DNA errors.

It has long been suggested that combining Chk1 inhibition with chemotherapy or radiotherapy should significantly enhance the anticancer effect of these therapies. This idea has serves as the basis for multiple pharmaceutical companies searching for potential Chk1 inhibitors that can effectively combine with chemotherapy in cancer therapy. To date, no Chk1 inhibitor has passed the clinical trial stage III . This led Dr. Zhang's team to look for alternative strategies for targeting Chk1 in cancer therapy.

Future research by Dr. Zhang and his team will consider two possible approaches to artificially activating Chk1 in cancer cells. One possibility is to use the gene therapy concept to deliver the active mutant form of Chk1 that the team discovered, into cancer cells. The other is to search for small molecules that can induce the same conformational change of Chk1, so that they can be delivered into cancer cells to activate Chk1 molecules. The consequence of either would be permanent cell proliferation inhibition and cancer.

All three authors of this study, Jingna Wang, Xiangzi Han and Youwei Zhang hold the title of Ph.D. and are members of the Department of Pharmacology, Case Western Reserve University School of Medicine, as well as members of the university's Case Comprehensive Cancer Center. Dr. Wang and Dr. Han are postdoctoral fellows. Dr. Zhang is an assistant professor. This study is published in Cancer Research. Support for the study comes from the National Cancer Institute at the National Institute of Health, Grants that supported this study are NCI R00CA126173 and R01CA163214.

http://www.wired.com/wiredscience/2012/08/pertussis-vax-effectiveness/

Is Childhood Pertussis Vaccine Less Effective Than We Thought?

Health authorities are beginning to open up a difficult topic: Whether the extraordinary ongoing epidemic of whooping cough, may be due in part to unexpected poor performance by the vaccine

By Maryn McKenna

Delicately and cautiously, health authorities in the United States and other countries are beginning to open up a difficult topic: Whether the extraordinary ongoing epidemic of whooping cough, the worst in more than 50 years, may be due in part to unexpected poor performance by the vaccine meant to prevent the disease.

That possibility, captured in several recent pieces of research - one published last night - is being raised so carefully because it might lead vaccine opponents to claim incorrectly that pertussis vaccination does not work. That fear contains a deep irony: The current vaccine, in use for about 20 years, replaced an older and more effective one that went out of use because vaccine critics charged it had too high a rate of side effects.

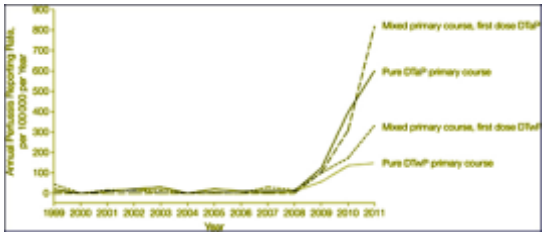
In the most recent research, a letter published Tuesday night in JAMA, researchers in Queensland, Australia examined the incidence of whooping cough in children who were born in 1998, the year in which that province began phasing out whole-cell pertussis vaccine (known as there as DTwP) in favor of less-reactive acellular vaccine (known as DTaP). Children who were born in that year and received a complete series of infant pertussis shots (at 2, 4 and 6 months) might have received all-whole cell, all-acellular, or a mix - and because of the excellent record-keeping of the state-based healthcare system, researchers were able to confirm which children received which shots. (NB: Queensland kids, like kids in the US, also receive boosters after the infant series, along with a final booster in their preteen years.)

The researchers were prompted to investigate because, like the US, Australia is enduring a ferocious pertussis epidemic. When they examined the disease history for 40,694 children whose vaccine history could be verified, they found 267 pertussis cases between 1999 and 2011. They said:

Children who received a 3-dose DTaP primary course had higher rates of pertussis than those who received a 3-dose DTwP primary course in the preepidemic and outbreak periods. Among those who received mixed courses, rates in the current epidemic were highest for children receiving DTaP as their first dose.

This pattern remained when looking at subgroups with 1 or 2 DTwP doses in the first year of life, although it did not reach statistical significance. Children who received a mixed course with DTwP as the initial dose had incidence rates that were between rates for the pure course DTwP and DTaP cohorts.

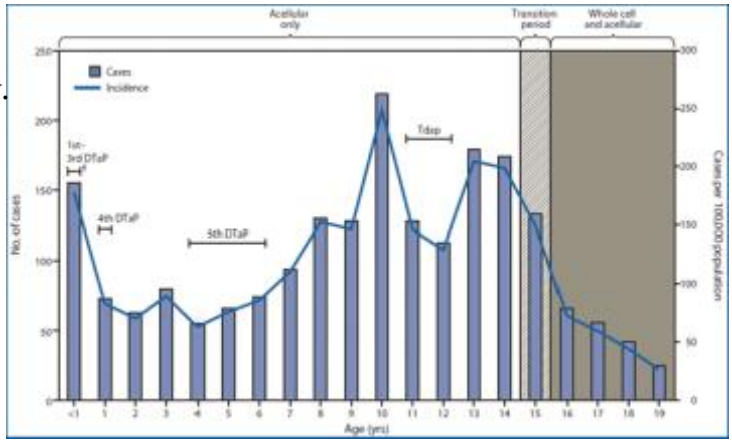
This figure from the paper graphs the different results:



Pertussis is cyclical, with peaks occurring every three to five years, but the authors (who come from the University of Queensland's Children's Medical Research Unit), say the effect they found persisted through both "pre-epidemic and outbreak" periods. They acknowledge it is possible that circulating strains of the whooping-cough bacterium, Bordetella pertussis, may have changed over the decade-plus since the vaccines were switched, but say the most reasonable explanation is that the immune protection conferred by DTaP does not last as long as that from the older vaccine.

This possibility has been raised before. Last fall, at the annual ICAAC infectious-disease meeting, physicians from Kaiser Permanente Medical Center in San Rafael, Calif. reported that they were seeing an unexpectedly high amount of pertussis in fully vaccinated pre-teens who had not yet received their final booster dose. Of 171 kids diagnosed by PCR as having pertussis in 2010, 132 were between 8 and 14. They said at the time that the rate of pertussis in the pre-teen group was "almost 20-fold" that of more recently vaccinated pre-schoolers, but subsided again in children older than 12 who received their last booster - and they questioned whether the DTaP vaccine's protection was waning earlier than expected and leaving the pre-teens vulnerable to infection. (The chart from their abstract is at right.)

Questioning the effectiveness of vaccines, in the midst of an epidemic and while they are under challenge from religious and "personal" exemptions, sounds like heresy - but in fact, the Centers for Disease Control and Prevention has raised the possibility just recently. The agency released a report July 20 on epidemics in Washington State (where cases are up 1,300 percent over last year) and and nationally. The report featured a widely circulated and dramatic graph of the epidemic curve - but it also included this less-reproduced graph, illustrating a difference in incidence between the whole-cell and acellular vaccine groups that resembles the data from Queensland:



Along with the graph, the report observed:

Acellular and whole-cell vaccines both have high efficacy during the first 2 years after vaccination, but recent changes in the epidemiology of pertussis in the United States strongly suggest diminished duration of protection afforded by childhood acellular vaccine (DTaP) compared with that of diphtheria and tetanus toxoids and whole-cell pertussis (DTwP) vaccine… Since the mid-2000s, the incidence of pertussis among children aged 7–10 years has increased. Moreover, the observed increase in risk by year of life from age 7–10 years suggests a cohort effect of increasing susceptibility as those children who exclusively received acellular vaccines continue to age.

In a media phone call that day, Dr. Anne Schuchat, director of the CDC's National Center for Immunization and Respiratory Diseases, went over the reason for the 20-year-old switch that may have fueled these rising rates of disease. She said:

Wholecell pertussis vaccines are widely used in many parts of the world. But in the U.S., we have not been using them since 1997… The wholecell pertussis vaccines had a fairly high rate of minor and short-term side effects like fever and pain and swelling at the injection site. Those were fairly common reactions. And the acellular pertussis vaccines have a lower rate of the fever and transient side effects. There were also rare, but serious neurologic adverse reactions, including chronic neurologic problems that occurred among children that recently received wholecell vaccines. Studies have not been consistent about whether the vaccine actually caused those chronic neurologic problems. Yet there was substantial public concern about

them and not just in the U.S., but in other countries. That led to a concerted effort to develop a vaccine with an improved safety profile.

Schuchat added:

In young children, we think that within a couple of years of vaccination the Dtap series is 95 percent protection. Five years later after the series, we think it wanes to 70 percent. That going down from 95 percent effectiveness to 70 percent may be why we see this increase in the older children or young teens.

There's an important footnote to that math, though. The vaccines confer protection on a certain percentage of the population that has been vaccinated - but if a substantial proportion of the population is not vaccinated, then what would otherwise be a small gap in the wall of herd immunity potentially can become a gaping hole. If the protection conferred by the childhood vaccination is waning unexpectedly early, then reinforcing vaccination at all ages - in childhood and also through adult boosters - becomes more important than ever.

Cites:

Sheridan SL, Ware RS, Grimwood K, Lambert SB. Number and Order of Whole Cell Pertussis Vaccines in Infancy and Disease Protection. JAMA. 2012;308(5):454-456. doi:10.1001/jama.2012.6364.

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http://news.discovery.com/earth/northernmost-coral-reef-found-120801.html#mkcpgn=rssnws1

World's Northernmost Coral Reef Discovered

The species and seascape are completely different from tropical reefs.

Content provided by Crystal Gammon, OurAmazingPlanet

Located off the coast of Japan's Tsushima Island at 34 degrees north latitude, the newly discovered reef is far different from other coral reefs. The corals live in water that is only 13 degrees Celsius in winter (55 degrees Fahrenheit).

When you think of coral reefs, you probably picture scuba divers gliding through warm, crystal-clear waters. And for the most part, you'd be right: more than 90 percent of the world's coral reefs are located in the tropics.

Now researchers in Japan have found what is - so far - the world's northernmost coral reef. Located off the coast of Tsushima Island at 34 degrees north latitude, the newly discovered reef is far different from other coral reefs and is 217 miles (350 kilometers) north of most others in the region.

Coral reefs discovered off the coast of Japan's Tsushima Island are the northernmost coral reef ever found on Earth.

Kaoru Sugihara

"Coral reefs have been believed to develop under warm-water settings - at least 18 degrees Celsius (64 degrees Fahrenheit) in winter. This setting is 13 degrees Celsius in winter (55 degrees Fahrenheit), which is unbelievably low," Hiroya Yamano, a researcher at Japan's National Institute for Environmental Studies, told OurAmazingPlanet. "The species, and thus seascape, is completely different from normal reefs."

A team led by Yamano found a similar reef off the coast of Japan's Iki Island in 2001, and until now, that reef was the planet's northernmost. The newfound Tsushima Island reef is 43 miles (70 km) north of the Iki Island reef. Yamano's team tracked down this reef after poring over old manuscripts and interviewing local residents. Their sleuthing paid off, and they eventually found the 4,300-year old reef in one of Tsushima's murky inner bays. "The water in this bay is basically turbid" - cloudy with lots of suspended particles - "and water temperatures are low, especially in winter," Yamano said.

Both of those qualities make life difficult for most corals. But this reef is composed of a genus of corals called Favia, a massive brown coral type. Most reefs are made up of corals from the genus Acropora, which can be a variety of colors and grow in branching or platelike polyps. Favia species tend to tolerate cold, turbid waters better than Acropora do, Yamano said.

So why did this reef start building itself in such an unfriendly environment? The team isn't sure, but Yamano thinks the Tsushima Warm Current, a stream of warm water flowing along the northwestern coast of Japan, probably helped transport coral larvae northward into the turbid inner bays of Iki and Tsushima islands. Yamano thinks there may be many more undiscovered reefs in similar settings throughout the region.

Changing species, changing climate?

Reefs like this one might help researchers measure ecosystem changes in warming oceans.

Although the Tsushima and Iki reefs both formed in very cold waters and predominantly house Favia coral species, Acropora corals have begun settling near the reefs over the last 20 years. Comparing coral species in older parts of the reef to newly arrived corals might help scientists determine how climate change and warming waters are affecting these reef ecosystems, Yamano said. His team's findings were published online July 12 in the journal Geology.

http://www.eurekalert.org/pub_releases/2012-08/aaon-aco072412.php

A cup of joe may help some Parkinson's disease symptoms

One of the first studies in humans to show that caffeine can help with movement symptoms for people who already have the disease

MINNEAPOLIS – While drinking caffeine each day does not appear to help improve sleepiness among people with Parkinson's disease, it may have a benefit in controlling movement, according to new research published in the August 1, 2012, online issue of Neurology®, the medical journal of the American Academy of Neurology .

"Studies have shown that people who use caffeine are less likely to develop Parkinson's disease, but this is one of the first studies in humans to show that caffeine can help with movement symptoms for people who already have the disease," said study author Ronald Postuma, MD, MSc, with McGill University in Montreal and the Research Institute of the McGill University Health Center. Postuma is also a member of the American Academy of Neurology.

For the study, 61 people with Parkinson's disease who showed symptoms of daytime sleepiness and some motor symptoms were given either a placebo pill or a pill with 100 milligrams of caffeine two times a day for three weeks, then 200 milligrams twice a day for three weeks, which was the equivalent of between two and four cups of coffee per day.

After six weeks, the half that took the caffeine supplements averaged a five-point improvement in Parkinson's severity ratings compared to those who didn't consume caffeine. "This is a modest improvement, but may be enough to provide benefit to patients. On the other hand, it may not be sufficient to explain the relationship between caffeine non-use and Parkinson's, since studies of the progression of Parkinson's symptoms early in the disease suggest that a five-point reduction would delay diagnosis by only six months," said Postuma.

The caffeine group also averaged a three-point improvement in the speed of movement and amount of stiffness compared to the placebo group. Caffeine did not appear to help improve daytime sleepiness and there were no changes in quality of life, depression or sleep quality in study participants.

"The study is especially interesting since caffeine seems to block a malfunctioning brain signal in Parkinson's disease and is so safe and inexpensive," said Michael Schwarzschild, MD, PhD, of Massachusetts General Hospital in Boston, who wrote an accompanying editorial. "Although the results do not suggest that caffeine should be used as a treatment in Parkinson's disease, they can be taken into consideration when people with Parkinson's are discussing their caffeine use with their neurologist." Schwarzschild is also a member of the American Academy of Neurology.

The study authors noted that the length of the study was short and that the effects of caffeine may lessen over time.

The study was supported by the Canadian Institute of Health Research and the Webster Foundation.

http://www.eurekalert.org/pub_releases/2012-08/acs-abf080112.php

Artificial butter flavoring ingredient linked to key Alzheimer's disease process

Evidence that the ingredient, diacetyl intensifies the damaging effects of an abnormal brain protein linked to Alzheimer's disease

A new study raises concern about chronic exposure of workers in industry to a food flavoring ingredient used to produce the distinctive buttery flavor and aroma of microwave popcorn, margarines, snack foods, candy, baked goods, pet foods and other products. It found evidence that the ingredient, diacetyl (DA), intensifies the damaging effects of an abnormal brain protein linked to Alzheimer's disease. The study appears in ACS' journal Chemical Research in Toxicology.

Robert Vince and colleagues Swati More and Ashish Vartak explain that DA has been the focus of much research recently because it is linked to respiratory and other problems in workers at microwave popcorn and food-flavoring factories. DA gives microwave popcorn its distinctive buttery taste and aroma. DA also forms naturally in fermented beverages such as beer, and gives some chardonnay wines a buttery taste. Vince's team realized that DA has an architecture similar to a substance that makes beta-amyloid proteins clump together in the brain - clumping being a hallmark of Alzheimer's disease. So they tested whether DA also could clump those proteins.

DA did increase the level of beta-amyloid clumping. At real-world occupational exposure levels, DA also enhanced beta-amyloid's toxic effects on nerve cells growing in the laboratory. Other lab experiments showed that DA easily penetrated the so-called "blood-brain barrier," which keeps many harmful substances from entering the brain. DA also stopped a protective protein called glyoxalase I from safeguarding nerve cells. "In light of the chronic exposure of industry workers to DA, this study raises the troubling possibility of long-term neurological toxicity mediated by DA," say the researchers.

The authors acknowledge funding from the Center for Drug Design (CDD) research endowment funds at the University of Minnesota, Minneapolis.

http://www.eurekalert.org/pub_releases/2012-08/bc-nsf072712.php

New study finds strong evidence of humans surviving rabies bites without treatment

First indication of people naturally protected against rabies found in remote Amazonian communities regularly exposed to vampire bats

Deerfield, IL Challenging conventional wisdom that rabies infections are 100 percent fatal unless immediately treated, scientists studying remote populations in the Peruvian Amazon at risk of rabies from vampire bats found 11 percent of those tested showed protection against the disease, with only one person reporting a prior rabies vaccination. Ten percent appear to have survived exposure to the virus without any medical intervention. The findings from investigators at the U.S. Centers for Disease Control and Prevention (CDC) were published today in the August 2012 issue of the American Journal of Tropical Medicine and Hygiene.

"The overwhelming majority of rabies exposures that proceed to infections are fatal. However, our results open the door to the idea that there may be some type of natural resistance or enhanced immune response in certain communities regularly exposed to the disease," said Amy Gilbert with the CDC's National Center for Emerging and Zoonotic Infectious Diseases, who is the paper's lead author. "This means there may be ways to develop effective treatments that can save lives in areas where rabies remains a persistent cause of death."

Rabies experts estimate the disease kills 55,000 people each year in Africa and Asia alone, and appears to be on the rise in China, the former Soviet Republics, southern Africa, and Central and South America. According to the CDC, in the United States, human deaths from rabies have declined over the past century from 100 annually to an average of two per year thanks to an aggressive campaign to vaccinate domestic animals against the disease.

In general, people who believe they may have been exposed to rabies are advised to immediately seek treatment which involves post-exposure prophylaxis (PEP) - a series of injections - to prevent the exposure from causing an active infection. These preventive treatments, when administered promptly, are 100 percent successful at preventing disease. Scientists have documented only a small number of individual cases, including one last year in California, in which an exposure to rabies proceeded to infection and the victim survived. Most of those survivors still required intensive medical attention, including one case in Wisconsin in which doctors induced a coma, though this approach has not been successful in most subsequent cases.

This CDC study was conducted in collaboration with the Peruvian Ministry of Health as part of a larger project to understand better bat-human interactions and its relation to rabies and emerging diseases that may be transmitted by bats. For their research, scientists traveled to two communities (Truenococha and Santa Marta) in a remote section of the Peruvian Amazon where outbreaks of fatal infections with rabies caused by bites from vampire bats - the most common "natural reservoir" for the disease in Latin America - have occurred regularly over the last two decades. They interviewed 92 people, 50 of whom reported previous bat bites. Blood samples were taken from 63 individuals and seven (11 percent) were found to have "rabies virus neutralizing antibodies."

One out of the seven individuals reported receiving a rabies vaccination - which generates antibodies to the rabies virus - but there was no evidence that the other six had received anti-rabies vaccine prior to the blood sampling or had sought out any medical attention for a bat bite, evidence that they had harbored the virus itself. The researchers acknowledged that they could not conclusively determine whether the antibodies were caused by an exposure to the virus that was somehow insufficient to produce disease. But they believe their evidence "suggests that (rabies virus) exposure is not invariably fatal to humans."

Gilbert said non-fatal exposures may happen more often than some think because "unless people have clinical symptoms of the disease they may not go to the hospital or clinic, particularly where access is limited."

"We all still agree that nearly everyone who is found to be experiencing clinical symptoms of rabies dies," Gilbert said. "But we may be missing cases from isolated high-risk areas where people are exposed to rabies virus and, for whatever reason, they don't develop disease."

In the Amazon region where the study was conducted - the Province Datem del Maranon in the Loreto Department of northern Peru - vampire bats, which live off of mammalian blood, regularly come out at night and prefer to feed on livestock. But in the absence of those food sources, they are known to seek out a meal from humans. They can use their extremely sharp teeth and the anticoagulant that naturally occurs in their saliva (appropriately referred to as "draculin") to feed on a sleeping person without awakening them. The rabies virus circulates extensively among vampire bat colonies in the region, and when an infected bat feeds, it passes along the virus to its host.

"This type of thorough and persistent scientific rabies investigation lends continued support to the belief that even the most dangerous of infectious diseases may be amenable to treatment," said James W. Kazura, MD noted infectious disease expert and president of the American Society of Tropical Medicine and Hygiene (ASTMH). "Continued investment of resources is essential for us to protect the health and well-being of innocent people whose lives and livelihoods are needlessly threatened by infectious diseases like rabies."

Gilbert and her colleagues hope their findings will prompt further studies in remote, at-risk communities to see if the results are replicated. In an editorial accompanying the study, Rodney E. Willoughby, a pediatric disease specialist at Children's Hospital of Wisconsin, said if it turns out there are distinct populations of people with "complete or relative resistance to rabies," there could be the potential to use whole genome sequencing to help develop new, life-saving treatments for rabies infections.

"Careful, respectful genetic study of these genetically unique populations may provide information on which pathways in human biochemistry and physiology promote resistance to human rabies," he wrote. "Equally important, knowing that there is a continuum of disease, even for infectious diseases like rabies, should push us harder to try for cures when confronted by so-called untreatable infectious diseases…."

Gilbert noted that the study was done as part of a larger public health effort to address a series of rabies outbreaks in the Amazon, where some health officials are now considering conducting pre-emptive vaccination campaigns in areas where risk of rabies is high and availability of medical care low. She said that while her study highlights people who appear to have survived an exposure to the virus, the fact remains that rabies outbreaks in small communities in the region have left tragic results. "These are very small villages and, when they witness ten people dying from what is a horrible disease, it is incredibly traumatic," Gilbert said. "We want to help raise awareness of the problem and try to develop a more proactive response."

http://www.eurekalert.org/pub_releases/2012-08/sumc-sii073012.php

Substance involved in Alzheimer's can reverse paralysis in mice with multiple sclerosis

A molecule widely assailed as the chief culprit in Alzheimer's disease unexpectedly reverses paralysis and inflammation in several distinct animal models of multiple sclerosis

STANFORD, Calif. — A molecule widely assailed as the chief culprit in Alzheimer's disease unexpectedly reverses paralysis and inflammation in several distinct animal models of a different disorder — multiple sclerosis, Stanford University School of Medicine researchers have found.

This surprising discovery, which will be reported in a study to be published online Aug. 1 as the cover feature in Science Translational Medicine, comes on the heels of the recent failure of a large-scale clinical trial aimed at slowing the progression of Alzheimer's disease by attempting to clear the much-maligned molecule, known as A-beta, from Alzheimer's patients' bloodstreams. While the findings are not necessarily applicable to the study of A-beta's role in the pathology of that disease, they may point to promising new avenues of treatment for multiple sclerosis.

The short protein snippet, or peptide, called A-beta (or beta-amyloid) is quite possibly the single most despised substance in all of brain research. It comes mainly in two versions differing slightly in their length and biochemical properties. A-beta is the chief component of the amyloid plaques that accumulate in the brains of Alzheimer's patients and serve as an identifying hallmark of the neurodegenerative disorder.

A-beta deposits also build up during the normal aging process and after brain injury. Concentrations of the peptide, along with those of the precursor protein from which it is carved, are found in multiple-sclerosis lesions as well, said Lawrence Steinman, MD, the new study's senior author. In a lab dish, A-beta is injurious to many types of cells. And when it is administered directly to the brain, A-beta is highly inflammatory.

Yet little is known about the physiological role A-beta actually plays in Alzheimer's — or in MS, said Steinman, a professor of neurology and neurological sciences and of pediatrics and a noted multiple-sclerosis researcher. He, first author Jacqueline Grant, PhD, and their colleagues set out to determine that role in the latter disease. (Grant was a graduate student in Steinman's group when the work was done.)

Multiple sclerosis, an inflammatory autoimmune disease, occurs when immune cells invade the brain and spinal cord and attack the insulating coatings of nerve cells' long, cable-like extensions called axons. Damage to these

coatings, composed largely of a fatty substance called myelin, disrupts the transmission of signals that ordinarily travel long distances down axons to junctions with other nerve cells. This signal disruption can cause blindness, loss of muscle control and difficulties with speech, thought and attention.

Previous research by Steinman, who is also the George A. Zimmerman Professor, and others showed that both A-beta and its precursor protein are found in MS lesions. In fact, the presence of these molecules along an axon's myelinated coating is an excellent marker of damage there.

Given the peptide's nefarious reputation, Steinman and his associates figured that A-beta was probably involved in some foul play with respect to MS. To find out, they relied on a mouse model that mimics several features of multiple sclerosis — including the autoimmune attack on myelinated sections of the brain that causes MS.

Steinman had, some years ago, employed just such a mouse model in research that ultimately led to the development of natalizumab (marketed as Tysabri), a highly potent MS drug. That early work proved that dialing down the activation and proliferation of immune cells located outside the central nervous system (which is what natalizumab does) could prevent those cells from infiltrating and damaging nerve cells in the CNS.

Knowing that immunological events outside the brain can have such an effect within it, the Stanford scientists were keen on seeing what would happen when they administered A-beta by injecting it into a mouse's belly, rather than directly to the brain. "We figured it would make it worse," Steinman said.

Surprisingly, the opposite happened. In mice whose immune systems had been "trained" to attack myelin, which typically results in paralysis, A-beta injections delivered before the onset of symptoms prevented or delayed the onset of paralysis. Even when the injections were given after the onset of symptoms, they significantly lessened the severity of, and in some cases reversed, the mice's paralysis.

Steinman asked Grant to repeat the experiment. She did, and got the same results.

His team then conducted similar experiments using a different mouse model: As before, they primed the mice's immune cells to attack myelin. But rather than test the effects of A-beta administration, the researchers harvested the immune cells about 10 days later, transferred them by injection to another group of mice that did not receive A-beta and then analyzed this latter group's response. The results mirrored those of the first set of experiments, proving that A-beta's moderating influence on the debilitating symptoms of the MS-like syndrome has nothing to do with A-beta's action within the brain itself, but instead is due to its effect on immune cells before they penetrate the brain.

Sophisticated laboratory tests showed that A-beta countered not only visible symptoms such as paralysis, but also the increase in certain inflammatory molecules that characterizes multiple-sclerosis flare-ups. "This is the first time A-beta has been shown to have anti-inflammatory properties," said Steinman.

Inspection of the central nervous systems of the mice with the MS-resembling syndrome showed fewer MS-like lesions in the brains and spinal cords of treated mice than in those not given A-beta. There was also no sign of increased Alzheimer's-like plaques in the A-beta-treated animals. "We weren't giving the mice Alzheimer's disease" by injecting A-beta into their bellies, said Grant. In addition, using an advanced cell-sorting method called flow cytometry, the investigators showed A-beta's strong effects on the immune system composition outside the brain. The numbers of immune cells called B cells were significantly diminished, while those of two other immune-cell subsets — myeloid cells and memory T-helper cells — increased.

"At this point we wanted to find out what would happen if we tried pushing A-beta levels down instead of up," Grant said. The researchers conducted a different set of experiments, this time in mice that lacked the gene for A-beta's precursor protein, so that they could produce neither the precursor nor A-beta. These mice, when treated with myelin-sensitized immune cells to induce the MS-like state, developed exacerbated symptoms and died faster and more frequently than normal mice who underwent the same regimen.

Lennart Mucke, MD, director of the Gladstone Institute of Neurological Disease in San Francisco and a veteran Alzheimer's researcher, noted that while A-beta's toxicity within the brain has been established beyond reasonable doubt, many substances made in the body can have vastly different functions under different circumstances.

"A-beta is made throughout our bodies all of the time. But even though it's been studied for decades, its normal function remains to be identified," said Mucke, who is familiar with Steinman's study but wasn't involved in it. "Most intriguing, to me, is this peptide's potential role in modulating immune activity outside the brain."

The fact that the protection apparently conferred by A-beta in the mouse model of multiple sclerosis doesn't require its delivery to the brain but, rather, can be attributed to its immune-suppressing effect in the body's peripheral tissues is likewise intriguing, suggested Steinman.

"There probably is a multiple-sclerosis drug in all this somewhere down the line," he said.

Additional Stanford co-authors were associate professor of neurology and neurological sciences Katrin Andreasson, MD; professor of genetics Leonore Herzenberg, DSc; emeritus professor of genetics Leonard Herzenberg, PhD; postdoctoral scholars Eliver Ghosn, PhD, Robert Axtell, PhD, Hedwich Kuipers, PhD, and Katja Herges, MD; and graduate student Nathan Woodling.

http://www.bbc.co.uk/news/science-environment-19077439

Palm trees 'grew on Antarctica'

Scientists drilling deep into the edge of modern Antarctica have pulled up proof that palm trees once grew there.

By Jason Palmer Science and technology reporter, BBC News

Analyses of pollen and spores and the remains of tiny creatures have given a climatic picture of the early Eocene period, about 53 million years ago. The study in Nature suggests Antarctic winter temperatures exceeded 10C, while summers may have reached 25C. Better knowledge of past "greenhouse" conditions will enhance guesses about the effects of increasing CO2 today.

The early Eocene - often referred to as the Eocene greenhouse - has been a subject of increasing interest in recent years as a "warm analogue" of the current Earth. The breakup of the last two supercontinents, Gondwana and Laurasia had begun, and Antarctica was not far from where it remains today, with Australia still to break off.

"There are two ways of looking at where we're going in the future," said a co-author of the study, James Bendle of the University of Glasgow. "One is using physics-based climate models; but increasingly we're using this 'back to the future' approach where we look through periods in the geological past that are similar to where we may be going in 10 years, or 20, or several hundred," he told BBC News.

The early Eocene was a period of atmospheric CO2 concentrations higher than the current 390 parts per million (ppm)- reaching at least 600ppm and possibly far higher. Global temperatures were on the order of 5C higher, and there was no sharp divide in temperature between the poles and the equator.

Drilling research carried out in recent years showed that the Arctic must have had a subtropical climate.

But the Antarctic presents a difficult challenge. Glaciation 34 million years ago wiped out much of the sediment that would give clues to past climate, and left kilometres of ice on top of what remains.

Now, the Integrated Ocean Drilling Program (IODP) has literally got to the bottom of what the Eocene Antarctic was like, dropping a drilling rig through 4km of water off Wilkes Land on Antarctica's eastern coast. The rig then drilled through 1km of sediment to return samples from the Eocene. With the sediment came pollen grains from palm trees and relatives of the modern baobab and macadamia. Crucially, they contained also the remnants of tiny single-celled organisms called Archaea.

The creatures' cell walls show subtle molecular changes that depend on the temperature of the soil surrounding them when they were alive. The structures are faithfully preserved after they die. They are, in essence, tiny buried thermometers from 53 million years ago. Together, the data suggest that even in the darkest period of Antarctic winter, the temperature did not drop below 10C; and summer daytime temperatures were in the 20Cs. The lowland coastal region sported palm trees, while slightly inland, hills were populated with beech trees and conifers.

Dr Bendle said that as an analogue of modern Earth, the Eocene represents heightened levels of CO2 that will not be reached any time soon, and may not be reached at all if CO2 emissions abate.

However, he said the results from the Eocene could help to shore up the computer models that are being used to estimate how sensitive climate is to the emissions that will certainly rise in the nearer term.

"It's a clearer picture we get of warm analogues through geological time," he said. "The more we get that information, the more it seems that the models we're using now are not overestimating the [climatic] change over the next few centuries, and they may be underestimating it. That's the essential message."

http://www.eurekalert.org/pub_releases/2012-08/uoc--acd073012.php

Alzheimer's cognitive decline slows in advanced age

The greatest risk factor for Alzheimer's disease (AD) is advancing age.

By age 85, the likelihood of developing the dreaded neurological disorder is roughly 50 percent. But researchers at the University of California, San Diego School of Medicine say AD hits hardest among the "younger elderly" – people in their 60s and 70s – who show faster rates of brain tissue loss and cognitive decline than AD patients 80 years and older. The findings, reported online in the August 2, 2012 issue of the journal PLOS One, have profound implications for both diagnosing AD – which currently afflicts an estimated 5.6 million Americans, a number projected to triple by 2050 – and efforts to find new treatments. There is no cure for AD and existing therapies do not slow or stop disease progression.

"One of the key features for the clinical determination of AD is its relentless progressive course," said Dominic Holland, PhD, a researcher at the Department of Neurosciences at UC San Diego and the study's first author. "Patients typically show marked deterioration year after year. If older patients are not showing the same deterioration from one year to the next, doctors may be hesitant to diagnose AD, and thus these patients may not receive appropriate care, which can be very important for their quality of life."

Holland and colleagues used imaging and biomarker data from participants in the Alzheimer's Disease Neuroimaging Initiative, a multi-institution effort coordinated at UC San Diego. They examined 723 people, ages 65 to 90 years, who were categorized as either cognitively normal, with mild cognitive impairment (an intermediate stage between normal, age-related cognitive decline and dementia) or suffering from full-blown AD.

"We found that younger elderly show higher rates of cognitive decline and faster rates of tissue loss in brain regions that are vulnerable during the early stages of AD," said Holland. "Additionally cerebrospinal fluid biomarker levels indicate a greater disease burden in younger than in older individuals."

Holland said it's not clear why AD is more aggressive among younger elderly.

"It may be that patients who show onset of dementia at an older age, and are declining slowly, have been declining at that rate for a long time," said co-author Linda McEvoy, PhD, associate professor of radiology. "But because of cognitive reserve or other still-unknown factors that provide 'resistance' against brain damage, clinical symptoms do not manifest till later age."

Another possibility, according to Holland, is that older patients may be suffering from mixed dementia – a combination of AD pathology and other neurological conditions. These patients might withstand the effects of AD until other adverse factors, such as brain lesions caused by cerebrovascular disease, take hold. At the moment, AD can only be diagnosed definitively by an autopsy. "So we do not yet know the underlying neuropathology of participants in this study," Holland said.

Clinical trials to find new treatments for AD may be impacted by the differing rates, researchers said. "Our results show that if clinical trials of candidate therapies predominately enroll older elderly, who show slower rates of change over time, the ability of a therapy to successfully slow disease progression may not be recognized, leading to failure of the clinical trial," said Holland. "Thus, it's critical to take into account age as a factor when enrolling subjects for AD clinical trials."

The obvious downside of the findings is that younger patients with AD lose more of their productive years to the disease, Holland noted. "The good news in all of this is that our results indicate those who survive into the later years before showing symptoms of AD will experience a less aggressive form of the disease."

Co-authors are Rahul S. Desikan, Department of Radiology, UCSD and Anders M. Dale, Departments of Neurosciences and Radiology, UCSD.

Funding for this research came, in part, from the National Institutes of Health (grants R01AG031224, R01AG22381, U54NS056883, P50NS22343 and P50MH08755); the National Institute on Aging (grant K01AG029218) and the National Institute of Biomedical Imaging and Bioengineering (grant T32EB005970).

http://www.eurekalert.org/pub_releases/2012-08/mu-iio073012.php

It's in our genes: Why women outlive men

Scientists are beginning to understand one of life's enduring mysteries - why women live, on average, longer than men.

Published today in Current Biology, research led by Monash University, describes how mutations to the DNA of the mitochondria can account for differences in the life expectancy of males and females. Mitochondria, which exist in almost all animal cells, are vital for life because they convert our food into the energy that powers the body.

Dr Damian Dowling and PhD student, Florencia Camus, both from the Monash School of Biological Sciences, worked with Dr David Clancy from Lancaster University to uncover differences in longevity and biological ageing across male and female fruit flies that carried mitochondria of different origins. They found that genetic variation across these mitochondria were reliable predictors of life expectancy in males, but not in females.

Dr Dowling said the results point to numerous mutations within mitochondrial DNA that affect how long males live, and the speed at which they age.

"Intriguingly, these same mutations have no effects on patterns of ageing in females. They only affect males," Dr Dowling said. "All animals possess mitochondria, and the tendency for females to outlive males is common to many different species. Our results therefore suggest that the mitochondrial mutations we have uncovered will generally cause faster male ageing across the animal kingdom."

The researchers said these mutations can be entirely attributed to a quirk in the way that mitochondrial genes are passed down from parents to offspring. "While children receive copies of most of their genes from both their mothers and fathers, they only receive mitochondrial genes from their mothers. This means that evolution's quality control process, known as natural selection, only screens the quality of mitochondrial genes in mothers," Dr Dowling said. "If a mitochondrial mutation occurs that harms fathers, but has no effect on mothers, this mutation will slip through the gaze of natural selection, unnoticed. Over thousands of generations, many such mutations have accumulated that harm only males, while leaving females unscathed."

The study builds on previous findings by Dr Dowling and his team that investigated the consequences of maternal inheritance of mitochondria in causing male infertility. "Together, our research shows that the mitochondria are hotspots for mutations affecting male health. What we seek to do now is investigate the genetic mechanisms that males might arm themselves with to nullify the effects of these harmful mutations and remain healthy," Dr Dowling said.

http://www.eurekalert.org/pub_releases/2012-08/ci-ftc080212.php

Fingering the culprit that polluted the Solar System

For decades it has been thought that a shock wave from a supernova explosion triggered the formation of our Solar System.

Washington, D.C. - According to this theory, the shock wave also injected material from the exploding star into a cloud of dust and gas, and the newly polluted cloud collapsed to form the Sun and its surrounding planets. New work from Carnegie's Alan Boss and Sandra Keiser provides the first fully three-dimensional (3-D) models for how this process could have happened. Their work will be published by The Astrophysical Journal Letters.

Traces of the supernova's pollution can be found in meteorites in the form of short-lived radioactive isotopes, or SLRIs. SLRIs—versions of elements with the same number of protons, but a different number of neutrons—found in primitive meteorites decay on time scales of millions of years and turn into different, so-called daughter, elements.

A million years may sound like a long time, but it is actually considered short when compared to other radioactive isotopes studied by geochemists and cosmochemists, which have half-lives measured in billions of years.

When scientists find the daughter elements distributed in telltale patterns in primitive meteorites, this means that the parent SLRIs had to be created just before the meteorites themselves were formed. This presents a timing problem, as the SLRIs must be formed in a supernova, injected into the presolar cloud, and trapped inside the meteoritic precursors, all in less than a million years.

The telltale patterns prove that the relevant daughter elements were not the ones that were injected. This is because the abundances of these daughters in different mineral phases in the meteorite are correlated with the abundances of a stable isotope of the parent element. Different elements have different chemical behaviors during the formation of these first solids, and the fact that the daughter elements correlate with the parent elements means that those daughters had to be derived from the decay of unstable parent elements after those solids were crystallized.

One of these SLRIs, iron-60, is only created in significant amounts by nuclear reactions in massive stars. The iron-60 must have come from a supernova, or from a giant star called an AGB star.

Boss and Keiser's previous modeling showed that it was likely that a supernova triggered our Solar System's formation, as AGB star shocks are too thick to inject the iron-60 into the cloud. Supernova shocks are hundreds of times thinner, leading to more efficient injection.

Now Boss and Keiser have extended those models to 3-D, so they can see the shock wave striking the gas cloud, compressing it and forming a parabolic shock front that envelopes the cloud, creating finger-like indentations in the cloud's surface. The fingers inject the SLRI pollution from the supernova. Less than 0.1 million years later, the cloud collapses and forms the core of the protostar that became the Sun and its surrounding planets.

The 3-D models show that only one or two fingers are likely to have caused the SLRI pollution found in primitive meteorites.

"The evidence leads us to believe that a supernova was indeed the culprit," said Boss. However, more detective work needs to be done: Boss and Keiser still need to find the combination of cloud and shock wave parameters that will line up perfectly with observations of exploding supernovae.

This research was supported by NASA. The software used in this work was, in part, developed by the DOE-supported ASC/Alliances Center for Astrophysical Thermonuclear Flashes a the University of Chicago

http://www.sciencedaily.com/releases/2012/08/120802092239.htm

Anacardic Acid Found to Rescue Certain ALS Abnormalities in Experimental Drug Screening Assay Using Motor Neurons from ALS Patient-Specific iPSCs

ScienceDaily - A research group at the Center for iPS Cell Research and Application (CiRA) at Japan's Kyoto University has successfully recapitulated amyotrophic lateral sclerosis (ALS)-associated abnormalities in motor neurons differentiated from induced pluripotent stem cells (iPSCs) obtained from patients with familial ALS, a late-onset, fatal disorder which is also known for Lou Gehrig's disease. In a drug screening assay using the disease model, the team further found that the chemical compound anacardic acid can rescue some ALS phenotypes in vitro.

In a study published online in Science Translational Medicine, Associate Professor Haruhisa Inoue and his team generated motor neurons from iPSCs derived from three ALS patients with mutations in Tar DNA-binding protein-43 (TDP-43). The motor neurons showed cellular phenotypes including vulnerability to stress, shorter neurites, and cytosolic aggregates similar to those seen in postmortem tissues from ALS patients. The team also found that TDP-43 mRNA was upregulated in the ALS motor neurons, which means that TDP-43 autoregulation was disturbed, and that TDP-43 protein in detergent-insoluble form aggregated with the splicing factor SNRPB2 in the nucleus, perturbing RNA metabolism. These findings shed light on the mechanism of disease onset.

Using the motor neurons as a disease model, the researchers discovered that the chemical compound anacardic acid can rescue the abnormal ALS motor neuron phenotypes. For example, when anacardic acid, a histone acetyltransferase inhibitor, was sprinkled on the motor neurons, TDP-43 mRNA expression was decreased, and the length of the neurites increased.

"Our work represents an initial stage of drug screening for ALS using patient-specific iPSCs. TDP-43 is not only relevant to familial ALS but also to sporadic ALS, which represents the majority of ALS cases," said Inoue, a principal investigator at CiRA who is also one of research directors for the CREST research program funded by the Japan Science and Technology Agency. "We will continue to work on ALS patient-specific iPSCs in order to help develop new drug seeds and candidates."

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http://www.eurekalert.org/pub_releases/2012-08/osu-pwa080312.php

People with allergies may have lower risk of brain tumors

New research adds to the growing body of evidence suggesting that there's a link between allergies and reduced risk of a serious type of cancer that starts in the brain.

COLUMBUS, Ohio - This study suggests the reduced risk is stronger among women than men, although men with certain allergy profiles also have a lower tumor risk. The study also strengthens scientists' belief that something about having allergies or a related factor lowers the risk for this cancer. Because these tumors, called glioma, have the potential to suppress the immune system to allow them to grow, researchers have never been sure whether allergies reduce cancer risk or if, before diagnosis, these tumors interfere with the hypersensitive immune response to allergens.

Scientists conducting this study were able to analyze stored blood samples that were taken from patients decades before they were diagnosed with glioma. Men and women whose blood samples contained allergy-related antibodies had an almost 50 percent lower risk of developing glioma 20 years later compared to people without signs of allergies. "This is our most important finding," said Judith Schwartzbaum, associate professor of epidemiology at Ohio State University and lead author of the study. "The longer before glioma diagnosis that the effect of allergies is present, the less likely it is that the tumor is suppressing allergies. Seeing this association so long before tumor diagnosis suggests that antibodies or some aspect of allergy is reducing tumor risk.

"It could be that in allergic people, higher levels of circulating antibodies may stimulate the immune system, and that could lower the risk of glioma," said Schwartzbaum, also an investigator in Ohio State's Comprehensive Cancer Center. "Absence of allergy is the strongest risk factor identified so far for this brain tumor, and there is still more to understand about how this association works." Many previous studies of the link between allergies and brain tumor risk have been based on self-reports of allergy history from patients

diagnosed with glioma. No previous studies have had access to blood samples collected longer than 20 years before tumor diagnosis.

The current study also suggested that women whose blood samples tested positive for specific allergy antibodies had at least a 50 percent lower risk for the most serious and common type of these tumors, called glioblastoma. This effect for specific antibodies was not seen in men. However, men who tested positive for both specific antibodies and antibodies of unknown function had a 20 percent lower risk of this tumor than did men who tested negative.

Glioblastomas constitute up to 60 percent of adult tumors starting in the brain in the United States, affecting an estimated 3 in 100,000 people. Patients who undergo surgery, radiation and chemotherapy survive, on average, for about one year, with fewer than a quarter of patients surviving up to two years and fewer than 10 percent surviving up to five years. The study is published online in the Journal of the National Cancer Institute.

Schwartzbaum and colleagues were granted access to specimens from the Janus Serum Bank in Norway. The bank contains samples collected from citizens during their annual medical evaluations or from volunteer blood donors for the last 40 years. Norway also has registered all new cases of cancer in the country since 1953, and personal identification numbers enable cross-referencing those cases with previously collected blood samples.

The researchers analyzed stored samples from 594 people who were diagnosed with glioma (including 374 diagnosed with glioblastoma) between 1974 and 2007. They matched these samples for date of blood collection, age and sex with 1,177 samples from people who were not diagnosed with glioma for comparison.

The researchers measured the blood samples for levels of two types of proteins called IgE, or immunoglobulin E. This is a class of antibodies produced by white blood cells that mediate immune responses to allergens. Two classes of IgE participate in the allergic response: allergen-specific IgE, which recognizes specific components of an allergen, and total IgE, which recognizes these components but also includes antibodies with unknown functions.

In each sample, the scientists determined whether the serum contained elevated levels of IgE specific to the most common allergens in Norway as well as total IgE. The specific respiratory allergens included dust mites; tree pollen and plants; cat, dog and horse dander; and mold. The researchers then conducted a statistical analysis to estimate the association between elevated concentrations of allergen-specific IgE and total IgE and the risk of developing glioma. Among women, testing positive for elevated levels of allergen-specific IgE was associated with a 54 percent decreased risk of glioblastoma compared to women who tested negative for allergen-specific IgE. The researchers did not see this association in men.

However, the relation between total IgE levels and glioma risk was not different for men and women, statistically speaking. For men and women combined, testing positive for elevated total IgE was linked to a 25 percent decreased risk of glioma compared with testing negative for total IgE.

The analysis for effects on glioblastoma risk alone suggested a similar decreased risk for both men and women combined whose samples tested positive for high levels of IgE, but the findings were considered borderline in terms of statistical significance, meaning the association could also be attributed to chance. "There is definitely a difference in the effect of allergen-specific IgE between men and women. And even results for total IgE suggest there still may be a difference between the sexes. The reason for this difference is unknown," Schwartzbaum said.

What the study does provide evidence for, however, is the likelihood that the immune systems of people with respiratory allergies could have a protective effect against this type of brain cancer. The ability to investigate this association over four decades between blood sampling and tumor diagnosis gave the researchers better insight into the relationship between allergies and tumor risk, Schwartzbaum said.

For example, a positive test for elevated concentrations of total IgE was associated with a 46 percent decreased risk for developing a glioma 20 years later compared to samples testing negative for elevated IgE, according to the analysis. That decreased risk was only about 25 percent in samples that tested positive for high levels of total IgE taken two to 15 years prior to diagnosis. "There may be a trend - the closer the samples get to the time of diagnosis, the less help the IgE is in decreasing the risk for glioma. However, if the tumor were suppressing allergy, we would expect to see a bigger difference in risk near the time of diagnosis," Schwartzbaum said.

Schwartzbaum plans to further analyze the serum samples for concentration of cytokines, which are chemical messengers that promote or suppress inflammation as part of the immune response, to see if these proteins have *a role in the relationship between elevated IgE levels and lowered tumor risk.*

This work was funded by the National Cancer Institute, the National Institutes of Health and a Research Enhancement and Assistance Program grant from Ohio State's Comprehensive Cancer Center.

Co-authors include Bo Ding, Anders Ahlbom and Maria Feychting of the Karolinska Institutet in Stockholm, Sweden; Tom Borge Johannesen and Tom Grimsrud of the Cancer Registry of Norway; Liv Osnes of Ulleval University Hospital in Oslo, Norway; and Linda Karavodin of Karavodin Preclinical Consulting in Encinitas, Calif.

http://bit.ly/RKsMO2

Two separate extinctions brought end to dinosaur era

The mass extinction that wiped out the dinosaurs 65 million years ago was almost unprecedented in its size.

17:04 03 August 2012 by Jeff Hecht

There may be a simple reason why three-quarters of Earth's species disappeared during the event – there were actually two extinctions at the end of the Cretaceous, each devastating species in distinct environments.

Famously, the dinosaurs met their end when a massive meteorite crashed into Mexico's Yucatán Peninsula around 65 million years ago. The extinction paved the way for the rapid evolutionary diversification of mammals.

But sceptics have long questioned whether the meteorite was solely responsible for the extinction. They point out that there were massive volcanic eruptions in India more than 100,000 years earlier, which triggered global warming that might have contributed to the species fatalities. But convincing evidence for those claims has proved elusive, so the impact has taken most of the blame.

A key problem has been finding sedimentary rocks that were formed at exactly the right time to capture all of the events that might have contributed to the extinction. The rocks need to contain plenty of fossils too, to reveal exactly when the various species disappeared. Thomas Tobin at the University of Washington in Seattle has just found rocks that fit the bill on Seymour Island, just off the Antarctic Peninsula. "It is really far south, so any climate changes are likely to be strongest there and have more biological effects," he says.

Tobin found two layers in the rocks, which formed in a shallow sea, where several species of shelled animals went extinct. One of the layers dates to the time of the impact, but the other layer is 40 metres below. Dating showed that the lower extinction occurred some 150,000 years before the meteorite hit – at the peak of the Indian eruptions. Tobin's team looked at isotopic ratios in the rock to work out the temperatures at the time: the first extinction followed a 7 °C rise in polar ocean temperatures – probably a result of global warming triggered by the Indian volcanism.

Comparable numbers of species in the region went extinct in each event. Surprisingly, though, the types of animals affected differed strikingly. "The stuff living at the [ocean] bottom died out during the [volcanic extinction event]," says Peter Ward, Tobin's thesis advisor and collaborator. That might be because the global warming triggered by the volcanic eruptions initially increased levels of biological activity in the oceans, but ultimately used up the oxygen dissolved in the water to create lethal anoxic conditions in deep water.

The later extinction, which is linked to the meteorite impact, wiped out creatures that lived in the surface waters.

The new data suggesting two distinct extinctions ties in with results of another new study. Gerta Keller of Princeton University and her team studied microfossils from the Bay of Bengal that lived during the end of the Cretaceous. The sea floor sediments in which they are preserved is interleaved with basalt from the massive Indian lava flows. Around half of the species went extinct during the initial volcanic eruptions, long before the meteorite impact. Here, however, it was the surface-dwelling organisms that were affected by the volcanism.

The case for multiple factors contributing to the extinction is adding up, says David Archibald, a vertebrate palaeontologist recently retired from San Diego State University, California, who was not involved in either study. "I'm not suggesting the [meteorite] impact didn't have tremendous effects, and it probably was necessary for the extinctions, but there were other things leading up to it," he says.

Journal reference: Tobin study: Palaeogeography, Palaeoclimatology, Palaeoecology DOI: 10.1016/j.palaeo.2012.06.029; Keller study: Earth and Planetary Science Letters, DOI: 10.1016/j.epsl.2012.06.021

http://www.bbc.co.uk/news/health-19112549

The power of intermittent fasting

Scientists are uncovering evidence that short periods of fasting, if properly controlled, could achieve a number of health benefits

Scientists are uncovering evidence that short periods of fasting, if properly controlled, could achieve a number of health benefits, as well as potentially helping the overweight, as Michael Mosley discovered.

I'd always thought of fasting as something unpleasant, with no obvious long term benefits. So when I was asked to make a documentary that would involve me going without food, I was not keen as I was sure I would not enjoy it. But the Horizon editor assured me there was great new science and that I might see some dramatic improvements to my body. So, of course, I said, "yes".

I am not strong-willed enough to diet over the long term, but I am extremely interested in the reasons why eating less might lead to increased life span, particularly as scientists think it may be possible to get the benefits without the pain.
How you age is powerfully shaped by your genes. But there's not much you can do about that. Calorie restriction, eating well but not much, is one of the few things that has been shown to extend life expectancy, at least in animals. We've known since the 1930s that mice put on a low-calorie, nutrient-rich diet live far longer. There is mounting evidence that the same is true in monkeys.

Growth hormone

The world record for extending life expectancy in a mammal is held by a new type of mouse which can expect to live an extra 40%, equivalent to a human living to 120 or even longer.
It has been genetically engineered so its body produces very low levels of a growth hormone called IGF-1, high levels of which seem to lead to accelerated ageing and age-related diseases, while low levels are protective.
A similar, but natural, genetic mutation has been found in humans with Laron syndrome, a rare condition that affects fewer than 350 people worldwide. The very low levels of IGF-1 their bodies produce means they are short, but this also seems to protect them against cancer and diabetes, two common age-related diseases.
The IGF-1 hormone (insulin-like growth factor) is one of the drivers which keep our bodies in go-go mode, with cells driven to reproduce. This is fine when you are growing, but not so good later in life.
But it turns out IGF-1 levels can be lowered by fasting. The reason seems to be that when our bodies no longer have access to food they switch from "growth mode" to "repair mode".
As levels of the IGF-1 hormone drop, a number of repair genes appear to get switched on according to ongoing research by Professor Valter Longo of the University of Southern California.

Intermittent fasting

One area of current research into diet is Alternate Day fasting (ADF), involving eating what you want one day, then a very restricted diet (fewer than 600 calories) the next, and most surprisingly, it does not seem to matter that much what you eat on non-fast days.
Dr Krista Varady of the University of Illinois at Chicago carried out an eight-week trial comparing two groups of overweight patients on ADF. "If you were sticking to your fast days, then in terms of cardiovascular disease risk, it didn't seem to matter if you were eating a high-fat or low-fat diet on your feed (non-fast) days," she said.
I decided I couldn't manage ADF, it was just too impractical. Instead I did an easier version, the so-called 5:2 diet. As the name implies you eat normally 5 days a week, then two days a week you eat 500 calories if you are a woman, or 600 calories, if you are a man.
There are no firm rules because so far there have been few proper human trials. I found that I could get through my fast days best if I had a light breakfast (scrambled eggs, thin slice of ham, lots of black tea, adding up to about 300 calories), lots of water and herbal tea during the day, then a light dinner (grilled fish with lots of vegetables) at night. On my feed days I ate what I normally do and felt no need to gorge.
I stuck to this diet for 5 weeks, during which time I lost nearly a stone and my blood markers, like IGF-1, glucose and cholesterol, improved. If I can sustain that, it will greatly reduce my risk of contracting age-related diseases like cancer and diabetes.
Current medical opinion is that the benefits of fasting are unproven and until there are more human studies it's better to eat at least 2000 calories a day. If you really want to fast then you should do it in a proper clinic or under medical supervision, because there are many people, such as pregnant women or diabetics on medication, for whom it could be dangerous. I was closely monitored throughout and found the 5:2 surprisingly easy. I will almost certainly continue doing it, albeit less often. Fasting, like eating, is best done in moderation.

http://phys.org/news/2012-08-climate-blame-extreme-nasa-scientist.htm

New NASA study links current extreme summer events to climate change

The weather-gone-crazy heat that has blistered parts of the world recently is so rare it can't be anything but man-made global warming, says a new statistical analysis from a top scientist.

The relentless, weather-gone-crazy type of heat that has blistered the United States and other parts of the world in recent years is so rare that it can't be anything but man-made global warming, says a new statistical analysis from a top government scientist.
The research by a man often called the "godfather of global warming" says that the likelihood of such temperatures occurring from the 1950s through the 1980s was rarer than 1 in 300. Now, the odds are closer to 1 in 10, according to the study by NASA scientist James Hansen. He says that statistically what's happening is not random or normal, but pure and simple climate change.

"This is not some scientific theory. We are now experiencing scientific fact," Hansen told The Associated Press in an interview.

Hansen is a scientist at NASA's Goddard Institute for Space Studies in New York and a professor at Columbia University. But he is also a strident activist who has called for government action to curb greenhouse gases for years. While his study was published online Saturday in the Proceedings of the National Academy of Science, it is unlikely to sway opinion among the remaining climate change skeptics. However, several climate scientists praised the new work. In a blunt departure from most climate research, Hansen's study — based on statistics, not the more typical climate modeling — blames these three heat waves purely on global warming:

—Last year's devastating Texas-Oklahoma drought.

—The 2010 heat waves in Russia and the Middle East, which led to thousands of deaths.

—The 2003 European heat wave blamed for tens of thousands of deaths, especially among the elderly in France.

The analysis was written before the current drought and record-breaking temperatures that have seared much of the United States this year. But Hansen believes this too is another prime example of global warming at its worst.

The new research makes the case for the severity of global warming in a different way than most scientific studies and uses simple math instead of relying on complex climate models or an understanding of atmospheric physics. It also doesn't bother with the usual caveats about individual weather events having numerous causes. The increase in the chance of extreme heat, drought and heavy downpours in certain regions is so huge that scientists should stop hemming and hawing, Hansen said. "This is happening often enough, over a big enough area that people can see it happening," he said.

Scientists have generally responded that it's impossible to say whether single events are caused by global warming, because of the influence of natural weather variability. However, that position has been shifting in recent months, as other studies too have concluded climate change is happening right before our eyes.

Hansen hopes his new study will shift people's thinking about climate change and goad governments into action. He wrote an op-ed piece that appeared online Friday in the Washington Post.

"There is still time to act and avoid a worsening climate, but we are wasting precious time," he wrote.

The science in Hansen's study is excellent "and reframes the question," said Andrew Weaver, a climate scientist at the University of Victoria in British Columbia who was a member of the Nobel Prize-winning international panel of climate scientists that issued a series of reports on global warming.

"Rather than say, 'Is this because of climate change?' That's the wrong question. What you can say is, 'How likely is this to have occurred with the absence of global warming?' It's so extraordinarily unlikely that it has to be due to global warming," Weaver said.

For years scientists have run complex computer models using combinations of various factors to see how likely a weather event would happen without global warming and with it. About 25 different aspects of climate change have been formally attributed to man-made greenhouse gases in dozens of formal studies. But these are generally broad and non-specific, such as more heat waves in some regions and heavy rainfall in others.

Another upcoming study by Kevin Trenberth, climate analysis chief at the National Center for Atmospheric Research, links the 2010 Russian heat wave to global warming by looking at the underlying weather that caused the heat wave. He called Hansen's paper an important one that helps communicate the problem.

But there is bound to be continued disagreement. Previous studies had been unable to link the two, and one by the National Oceanic and Atmospheric Administration concluded that the Russian drought, which also led to devastating wildfires, was not related to global warming.

White House science adviser John Holdren praised the paper's findings in a statement. But he also said it is true that scientists can't blame single events on global warming: "This work, which finds that extremely hot summers are over 10 times more common than they used to be, reinforces many other lines of evidence showing that climate change is occurring and that it is harmful."

Skeptical scientist John Christy of the University of Alabama at Huntsville said Hansen shouldn't have compared recent years to the 1950s-1980s time period because he said that was a quiet time for extremes.

But Derek Arndt, director of climate monitoring for the federal government's National Climatic Data Center, said that range is a fair one and often used because it is the "golden era" for good statistics.

Granger Morgan, head of engineering and public policy at Carnegie Mellon University, called Hansen's study "an important next step in what I expect will be a growing set of statistically-based arguments."

In a landmark 1988 study, Hansen predicted that if greenhouse gas emissions continue, which they have, Washington, D.C., would have about nine days each year of 95 degrees or warmer in the decade of the 2010s.

So far this year, with about four more weeks of summer, the city has had 23 days with 95 degrees or hotter temperatures.
Hansen says now he underestimated how bad things would get. And while he hopes this will spur action including a tax on the burning of fossil fuels, which emit carbon dioxide, a key greenhouse gas, others doubt it. Science policy expert Roger Pielke Jr. of the University of Colorado said Hansen clearly doesn't understand social science, thinking a study like his could spur action. Just because something ought to happen, doesn't mean it will, he said.
In an email, he wrote: "Hansen is pursuing a deeply flawed model of policy change, one that will prove ineffectual and with its most lasting consequence a further politicization of climate science (if that is possible!)."
More information: "Perception of climate change," by James Hansen, Makiko Sato, and Reto Ruedy, PNAS, 2012.

http://www.bbc.co.uk/news/health-19111700

Chemo 'undermines itself' through rogue response

Chemotherapy can undermine itself by causing a rogue response in healthy cells, which could explain why people become resistant, a study suggests.

The treatment loses effectiveness for a significant number of patients with secondary cancers. Writing in Nature Medicine, US experts said chemo causes wound-healing cells around tumours to make a protein that helps the cancer resist treatment. A UK expert said the next step would be to find a way to block this effect.
Around 90% of patients with solid cancers, such as breast, prostate, lung and colon, that spread - metastatic disease - develop resistance to chemotherapy. Treatment is usually given at intervals, so that the body is not overwhelmed by its toxicity. But that allows time for tumour cells to recover and develop resistance.
In this study, by researchers at the Fred Hutchinson Cancer Research Center in Seattle looked at fibroblast cells, which normally play a critical role in wound healing and the production of collagen, the main component of connective tissue such as tendons. But chemotherapy causes DNA damage that causes the fibroblasts to produce up to 30 times more of a protein called WNT16B than they should. The protein fuels cancer cells to grow and invade surrounding tissue - and to resist chemotherapy.

Success v failure

It was already known that the protein was involved in the development of cancers - but not in treatment resistance. The researchers hope their findings will help find a way to stop this response, and improve the effectiveness of therapy.
Peter Nelson, who led the research, said: "Cancer therapies are increasingly evolving to be very specific, targeting key molecular engines that drive the cancer rather than more generic vulnerabilities, such as damaging DNA. "Our findings indicate that the tumour microenvironment also can influence the success or failure of these more precise therapies."
Prof Fran Balkwill, a Cancer Research UK expert on the microenvironment around tumours, said: "This work fits with other research showing that cancer treatments don't just affect cancer cells, but can also target cells in and around tumours. "Sometimes this can be good - for instance, chemotherapy can stimulate surrounding healthy immune cells to attack tumours. "But this work confirms that healthy cells surrounding the tumour can also help the tumour to become resistant to treatment. "The next step is to find ways to target these resistance mechanisms to help make chemotherapy more effective."

http://www.eurekalert.org/pub_releases/2012-08/cu-pdr080112.php

Pupil dilation reveals sexual orientation in new Cornell study

There is a popular belief that sexual orientation can be revealed by pupil dilation to attractive people, yet until now there was no scientific evidence.

ITHACA, N.Y. - For the first time, researchers at Cornell University used a specialized infrared lens to measure pupillary changes to participants watching erotic videos. Pupils were highly telling: they widened most to videos of people who participants found attractive, thereby revealing where they were on the sexual spectrum from heterosexual to homosexual. The findings were published August 3 in the scientific journal PLoS ONE (http://dx.plos.org/10.1371/journal.pone.0040256).
Previous research explored these mechanisms either by simply asking people about their sexuality, or by using physiological measures such as assessing their genital arousal. These methods, however, come with substantial problems.
"We wanted to find an alternative measure that would be an automatic indication of sexual orientation, but without being as invasive as previous measures. Pupillary responses are exactly that," says Gerulf Rieger, lead author and research fellow at Cornell. "With this new technology we are able to explore sexual orientation of

people who would never participate in a study on genital arousal, such as people from traditional cultures. This will give us a much better understanding how sexuality is expressed across the planet."
The new Cornell study adds considerably more to the field of sexuality research than merely a novel measure. As expected, heterosexual men showed strong pupillary responses to sexual videos of women, and little to men; heterosexual women, however, showed pupillary responses to both sexes. This result confirms previous research suggesting that women have a very different type of sexuality than men.
Moreover, the new study feeds into a long-lasting debate on male bisexuality. Previous notions were that most bisexual men do not base their sexual identity on their physiological sexual arousal but on romantic and identity issues. Contrary to this claim, bisexual men in the new study showed substantial pupil dilations to sexual videos of both men and women.
"We can now finally argue that a flexible sexual desire is not simply restricted to women – some men have it, too, and it is reflected in their pupils," says Ritch C. Savin-Williams, co-author and professor in Human Development at Cornell. "In fact, not even a division into 'straight,' 'bi,' and 'gay' tells the full story. Men who identity as 'mostly straight' really exist both in their identity and their pupil response; they are more aroused to males than straight men, but much less so than both bisexual and gay men," Savin-Williams notes.
The researchers are confident that their new measure will aid in understanding these groups better and point to a range of sexualities that has been ignored in previous research.

http://phys.org/news/2012-08-star-trak-august.html

STAR TRAK for August 2012

The annual Perseid meteor shower, which will peak on Aug. 11-12, is one of the most popular every year

The annual Perseid meteor shower, which will peak on Aug. 11-12, is one of the most popular every year because it happens on warm summer nights, when gazing at the starry sky is always enjoyable. In a clear, dark sky there may be as many as 60 bright meteors per hour, some with smoke trails that last several seconds after the meteor has vanished. Start observing around midnight local daylight time. A crescent moon will rise around 1 a.m., but it won't have much effect.
The Perseids will be visible for most of August, though there will be fewer meteors to see the further from the peak date you watch. If the peak is hidden by clouds, for example, try looking for meteors again as soon as the night sky is clear. To minimize the effect of local light pollution, which can obscure as many as half of the meteors, try to avoid artificial lights. Face east if you have a clear view in that direction, and look about half way up the sky from the horizon. You won't need binoculars or a telescope because the meteors move much too fast for those. The chances of seeing a fireball will be greatest near dawn, when Earth will be moving head on into the meteor stream.
The Perseids may appear anywhere in the sky, but they will seem to originate from a point called the radiant in the constellation Perseus, which gives the meteors their name. The higher the radiant is above the northeastern horizon, the more meteors will be visible. Perseus is just north of the W-shaped constellation Cassiopeia in the Milky Way, with the bright star Capella and the Pleiades star cluster below it. Meteors near the radiant will have short trails because we see them nearly end on, while those far from the radiant will look longer because they are seen from the side. A computer simulation of meteors streaking from the Perseid shower's radiant can be seen at www.shadowandsubstance.com/ .
Most meteor showers happen when Earth crosses the orbit of a comet; the Perseids come from Comet Swift Tuttle. The meteors are caused by particles released from the comet's nucleus and left behind in space. As Earth plows through this stream of debris, ranging in size from sand grains to pebbles, each particle slams into our atmosphere at a speed of more than 50 kilometers per second and burns up almost instantly from friction with air molecules. The resulting heat momentarily creates a streak of glowing air that we see as a meteor (sometimes called a "shooting star" or "falling star"). All of this happens about 50 miles above the ground, regardless of how close some meteors may appear. More information about the Perseids and other meteor showers is available at www.skyandtelescope.com/observing/objects/meteors.

Planets

At the beginning of August, Saturn and Mars will be low in the west-southwest an hour after sunset, along with the bright star Spica. The two planets will gradually approach each other as the days pass, and on Aug. 7 the three objects will form an equilateral triangle. Golden-yellow Saturn will be distinct from red-orange Mars, while blue-white Spica will contrast with both planets. Mars will pass between Saturn and Spica on Aug. 13 and 14, with the three forming a nearly straight line. On Aug. 21, they will make another equilateral triangle

with Mars on the opposite side. The trio will set within two hours after the sun. On Aug. 5-6, NASA's Curiosity rover is scheduled to touch down on the surface of Mars.
Saturn's rings will be tilted 14 degrees to our line of sight in mid-August. Any telescope will show Saturn's biggest moon, Titan, which will be south of the planet on Aug. 8 and 24, and north of it on Aug. 16.
Jupiter will rise around 2 a.m. local time in early August just north of the bright orange star Aldebaran in the constellation Taurus the Bull. The planet will slowly cross Taurus during August, rising shortly before midnight by the end of the month. When it is reasonably high above the eastern horizon in early morning twilight, it will be a splendid sight in a telescope.
Even brighter than Jupiter will be Venus when it rises more than three hours before the sun and dominates the morning sky. The brilliant planet will reach its greatest elongation from the sun on Aug. 15, a fine time for viewing it with a telescope.
Mercury will be far to the lower left (north) of Venus and difficult to find until it gets higher. On Aug. 16, the planet will appear 10 degrees above the eastern horizon a half hour before sunrise.

Light pollution

If you look at the constellation Cassiopeia on a clear summer night, and you can't see the Milky Way sprawling high across the sky from the northern to the southern horizon, then your sky has significant light pollution, which is the case for about two thirds of the world's population. See www.darksky.org/ for information on this dimming of the night sky caused by excessive artificial lighting, much of which is wasted.

Moon phases

The moon will be full on Aug. 1, at third quarter on Aug. 9, new on Aug. 17, at first quarter on Aug. 24 and full again (a "blue moon") on Aug. 31. *Provided by Indiana University*

http://phys.org/news/2012-08-greed-middle-ages.html

Greed was different in the Middle Ages, researcher says

Greed was different in the Middle Ages, says Stanford's Laura Stokes

Phys.org - Surveys of the carnage of the American financial crisis that began in 2008 have revealed the potent allure of personal gain above all else.
But greed hasn't always been popular in Western societies.
Stanford historian Laura Stokes is uncovering how attitudes toward "acceptable greed" have done a turnaround in the past 500 years.
Self-serving behavior deemed necessary on Wall Street today might have been despised in medieval Europe. One might even have been murdered for using wealth as a justification for circumventing societal norms.
Capitalism, Stokes has found, managed to flourish in the intensely community-conscious culture of medieval times. Men of business successfully built financial empires based on trade and credit, even though unbridled greed was universally condemned.

A depiction of a medieval market. While businessmen in the Middle Ages did amass personal fortunes, open greed was unacceptable to the community and could even lead to murder. **Image: Wikimedia Commons**

The question that perplexes Stokes, an assistant professor of history, is how such men could be admired by their peers, when greed was frowned upon.
In short, blatantly selfish economic behavior was simply unacceptable. In describing the contradiction between present-day business attitudes and a medieval mindset, Stokes said, "A medieval businessman would surely be impressed by the successes of his modern descendants, but he would also despise them as men without honor or virtue."
Stokes, a historian of early modern Europe, began her research when she came across unusually extensive documentation on financial disputes from the medieval era. While poring through the documents at the Staatsarchiv Basel-Stadt (an important archive of the city of Basel), she was intrigued by the amount of text dedicated to preserving every detail of these interactions – down to specific "he said, she said" conversations.
It was compelling, she explained, because even when people were relating financial experiences that happened 20 years prior, "They were offering quoted speech as if the events had happened the day before."
Excited by this detail, Stokes delved more deeply into the records to look for a pattern in the language people used to describe their financial disputes. In examples of court depositions, she found that people emphasized the collective damage done to the community over their own losses.

In a 16th-century quarrel between cousins, "one man criticized another's greedy behavior, saying, 'Cousin, cousin, you've acted poorly and committed injustice,' " Stokes said.

The story of Klein Hans Fisher, a Swiss man who owed a massive debt on his mortgage, highlights the difference between our modern financial mindset and the medieval one.

As the court records show, a wealthy businessman in Lucerne had issued Fisher the mortgage in the late 16th century. Some time later, Fisher fell behind on his payments. Rather than seize the land from Fisher, the businessman, who was also the Lord Mayor Badmer, gave it to Fisher's sons and worked out a rental agreement with them so they would keep the land in the family.

The records indicate that Fisher visited the land at harvest time and took the "rent" payment due to Lord Badmer. As Stokes explained, "Hans Fisher visits the land and takes the excess harvest for himself, along with some farming equipment, leaving his sons with no rent to pay Lord Badmer."

But Badmer does not exercise his legal right to repossess the land – that would have been unacceptable behavior. "Not only farmers, but also the rich men in the city understood that land belonged to families in ways that debt could not erase," Stokes said.

In another, more dramatic, example of the community rejection of selfish business practices, murder was seen as the only response severe enough to deal with a pompous businessman, Uly Mörnach. He was a property owner who insisted he had the power and the right to do what he wanted with his property. As Stokes found by sifting through the archives of the city of Basel, "He insisted on a kind of individualistic … perspective on his own life."

Although his impressive property holdings might be admired in today's culture, he was seen as downright despicable by his medieval peers for the way he threw his financial weight around. In one instance, Stokes found, he beat an old woman when he discovered her taking water from his meadow. When the woman pressed charges, Mörnach lied about the matter in court, and laughed about it later to his friends.

In medieval society, his disregard for the rules of social responsibility and the value of community honor was a misstep that disturbed his neighbors deeply, so much so that they collectively conspired to murder him – with many of them escaping legal repercussions.

Stokes has found religious studies to be an invaluable area of academic insight into understanding patterns of social attitudes in 16th-century Europe. "Theology," Stokes said, "is actually fundamentally important to me to understand greed and the crimes associated with it later, in terms of sin and moral rights."

Stokes, however, is careful to note that social attitudes and religious attitudes, while related, are by no means the same. Her primary interest and material for the exploration of the history of greed is not the religious condemnation of the practice as much as it is about the shared moral code that she has found across religious beliefs in collective communities.

"The heart of the arguments of the people I'm researching," she explained, "are socially indigenous value sets – not from an outside institution."

And, while quite different, "these value sets are present in today's society," said Stokes. Despite a heavy emphasis on greed in modern business culture, we still value social responsibility. As Stokes pointed out, "We admire most our great philanthropists who can balance both." *Provided by Stanford University*

Nasa's Curiosity rover successfully lands on Mars

The US space agency has just landed a huge new robot rover on Mars.

By Jonathan Amos Science correspondent, BBC News, Pasadena

The one-tonne vehicle, known as Curiosity, was reported to have landed in a deep crater near the planet's equator at 06:32 BST (05:32 GMT). It will now embark on a mission of at least two years to look for evidence that Mars may once have supported life.

A signal confirming the rover was on the ground safely was relayed to Earth via Nasa's Odyssey satellite, which is in orbit around the Red Planet. The success was greeted with a roar of approval here at mission control at the Jet Propulsion Laboratory (JPL) in Pasadena, California.

The reaction from the Nasa control room as the rover landed

Within minutes, the robot was returning its first low-resolution images - showing us its wheels and views to the horizon. A first colour image of Curiosity's surroundings should be returned in the next couple of days.

Engineers and scientists who have worked on this project for the best part of 10 years punched the air and hugged each other. The rover's Twitter feed announced: "I'm safely on the surface of Mars. GALE CRATER I AM IN YOU!!!"

The descent through the atmosphere after a 570-million-km journey from Earth had been billed as the "seven minutes of terror" - the time it would take to complete a series of high-risk, automated manoeuvres that would slow the rover from an entry speed of 20,000km/h to allow its wheels to set down softly.

The first pictures from Mars began to be fed back immediately; high-resolution images will come later

Analysis David Shukman Science editor, BBC News

The day I watched Curiosity being built in a clean room at Nasa's Jet Propulsion Laboratory in Pasadena last year, the rover's six wheels were lying on one work bench while the chassis stood on another and it was hard to believe the white-suited engineers could make sense of the maze of tubes and cabling. But what they've created now stands on the red soil of Mars - and it's in one piece. In the hallway of a JPL building we were shown a full-size replica. Walking around it made me realise something difficult to grasp from the pictures and video: this is a beast of a machine, a kind of cosmic Humvee with instruments instead of weapons.

Sometimes Nasa public relations can appear bragging. Today it feels justified. Curiosity is all set to discover something remarkable about our strangest neighbour.

The Curiosity team had to wait 13 tense minutes for the signals from Odyssey and the lander to make their way back to Earth.

Data suggested the vehicle had hit the surface of Mars at a gentle 0.6m/s.

"It looked at least with my eyeball that we landed in a nice flat spot. Beautiful," said Adam Steltzner, who led the descent operation.

The JPL director, Charles Elachi, added: "Tonight was a great drama that was played. I felt like I was in an adventure movie but I kept telling myself this is real; and what a fantastic demonstration of what our nation and our agency can do."

That sense of national pride was picked up by US President Barack Obama's chief science adviser, John Holdren.

"Landing the Mars Science Laboratory rover Curiosity on the Red Planet was by any measure the most challenging mission ever attempted in the history of planetary exploration," he said. "And if anyone has been harbouring doubts about the status of US leadership in space, well there's a one tonne automobile-sized piece of American ingenuity sitting on the surface of the Red Planet right now."

This is the fourth rover Nasa has put on Mars, but its scale and sophistication dwarf all previous projects. Its biggest instrument alone is nearly four times the mass of the very first robot rover deployed on the planet back in 1997.

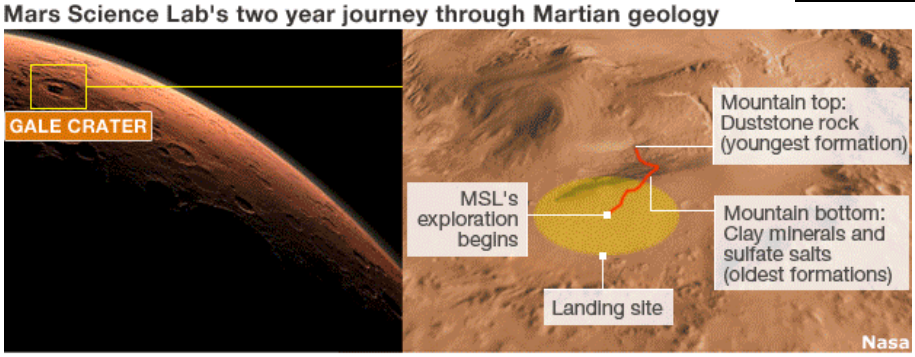
Curiosity has been sent to investigate the central mountain inside Gale Crater that is more than 5km high.

It will climb the rise, and, as it does so, study rocks that were laid down billions of years ago in the presence of liquid water. The vehicle will be scouring Mount Sharp in the crater's centre looking for evidence that past environments could have favoured microbial life.
It is a region that Curiosity project scientist John Grotzinger told the BBC's Horizon programme reads like a "book about the early environmental history of Mars".
Scientists warn, however, that this will be a slow mission - Curiosity is in no hurry. For one thing, the rover has a plutonium battery that should give it far greater longevity than the solar-panelled power systems fitted to previous vehicles.
"People have got to realise this mission will be different," commented Steve Squyres, the lead scientist of the Opportunity and Spirit rovers put on the surface in 2004.
"When we landed we only thought we'd get 30 sols (Martian days) on the surface, so we had to hit the ground running. Curiosity has plenty of time," he told the BBC.
Initially, the rover is funded for two Earth years of operations. But many expect this mission to roll and roll for perhaps a decade or more.

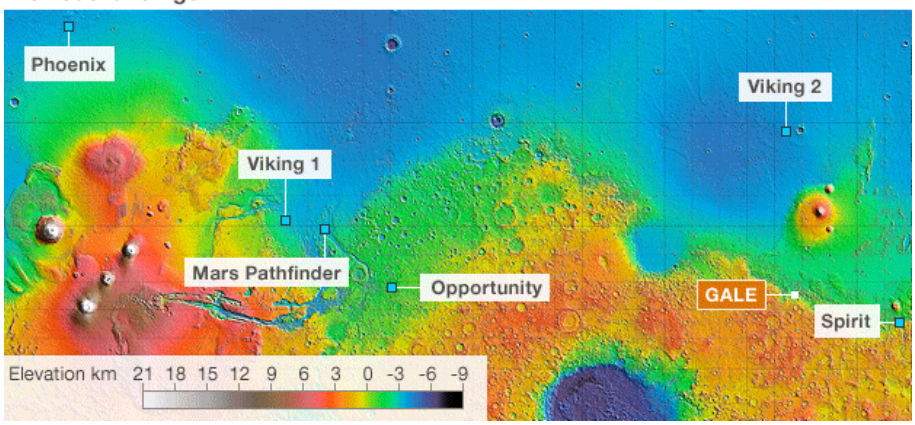
Curiosity - Mars Science Laboratory

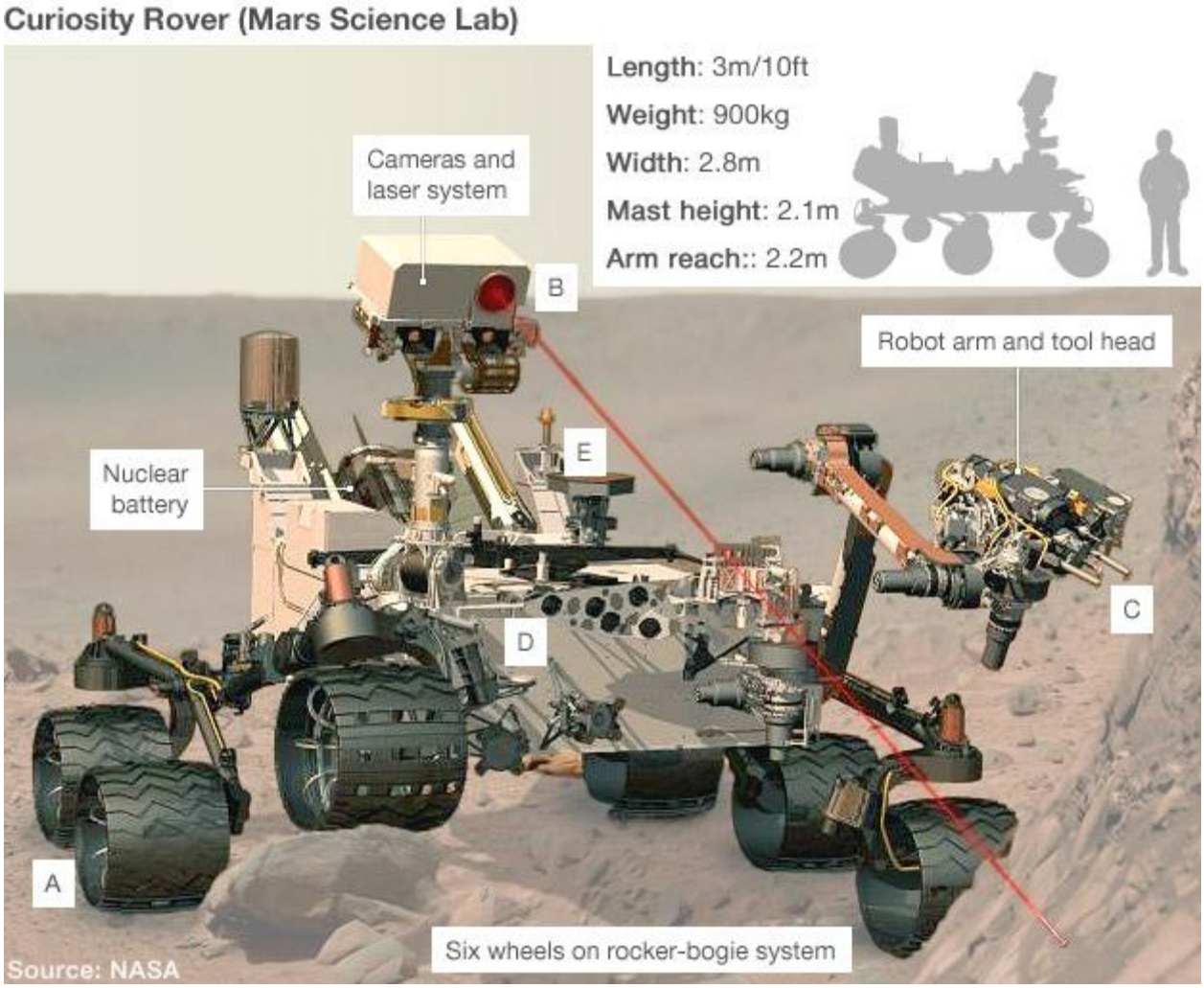
- ***Mission goal is to determine whether Mars has ever had the conditions to support life***
- ***Project costed at $2.5bn; will see initial surface operations lasting two Earth years***
- ***Onboard plutonium generators will deliver heat and electricity for at least 14 years***
- ***75kg science payload more than 10 times as massive as those of earlier US Mars rovers***
- ***Equipped with tools to brush and drill into rocks, to scoop up, sort and sieve samples***
- ***Variety of analytical techniques to discern chemistry in rocks, soil and atmosphere***
- ***Will try to make first definitive identification of organic (carbon rich) compounds***
- ***Even carries a laser to zap rocks; beam will identify atomic elements in rocks***

Mars Science Lab's two year journey through Martian geology



Previous landings





(A) Curiosity will trundle around its landing site looking for interesting rock features to study. Its top speed is about 4cm/s
(B) This mission has 17 cameras. They will identify particular targets, and a laser will zap those rocks to probe their chemistry
(C) If the signal is significant, Curiosity will swing over instruments on its arm for close-up investigation. These include a microscope
(D) Samples drilled from rock, or scooped from the soil, can be delivered to two hi-tech analysis labs inside the rover body
(E) The results are sent to Earth through antennas on the rover deck. Return commands tell the rover where it should drive next