

## Computer with 'Schizophrenia'!



TELLTALE ROCK

COMPUTER networks that can't forget fast enough can show symptoms of a kind of virtual schizophrenia, giving researchers further clues to the inner workings of schizophrenic brains, researchers at The University of Texas at Austin and Yale University have found.

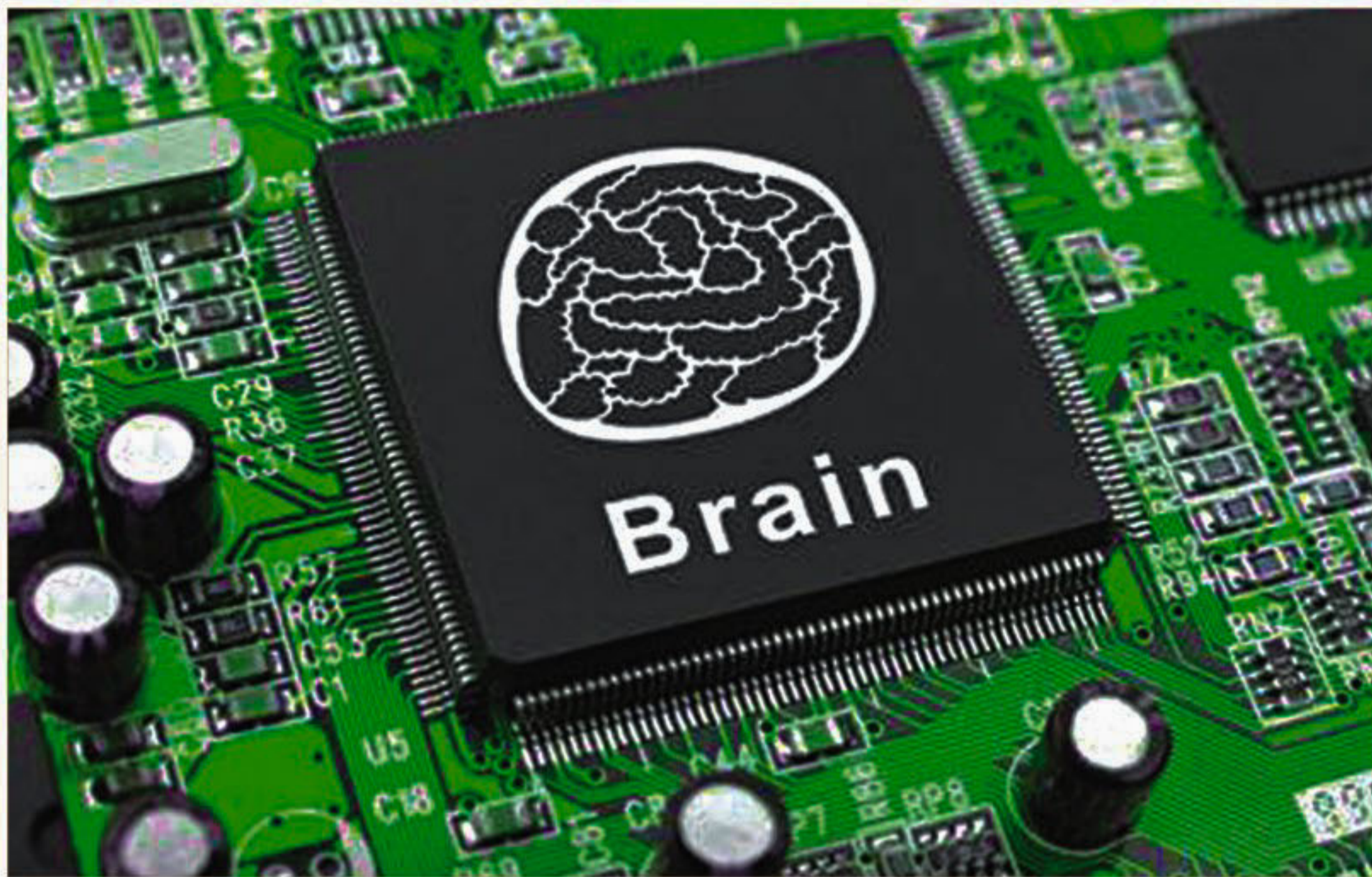
The researchers used a virtual computer model, or "neural network," to simulate the excessive release of dopamine in the brain. They found that the network recalled memories in a distinctly schizophrenic-like fashion.

Their results were published in April in Biological Psychiatry.

"The hypothesis is that dopamine encodes the importance-of experience," says Uli Grasemann, a graduate student in the Department of Computer Science at The University of Texas at Austin. "When there's too much dopamine, it leads to exaggerated salience, and the brain ends up learning from things that it shouldn't be learning from."

The results bolster a hypothesis known in schizophrenia circles as the hyperlearning hypothesis, which posits that people suffering from schizophrenia have brains that lose the ability to forget or ignore as much as they normally would. Without forgetting, they lose the ability to extract what's meaningful out of the immensity of stimuli the brain encounters. They start making connections that aren't real, or drowning in a sea of so many connections they lose the ability to stitch together any kind of coherent story.

The neural network used by



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Grasemann and his adviser, Professor Risto Miikkulainen, is called DISCERN. Designed by Miikkulainen, DISCERN is able to learn natural language. In this study it was used to simulate what happens to language as the result of eight different types of neurological dysfunction. The results of the simulations were compared by Ralph Hoffman, professor of psychiatry at the Yale School of Medicine, to what he saw when studying human schizophrenics.

In order to model the process, Grasemann and Miikkulainen

began by teaching a series of simple stories to DISCERN. The stories were assimilated into DISCERN's memory in much the way the human brain stores information—not as distinct units, but as statistical relationships of words, sentences, scripts and stories.

"With neural networks, you basically train them by showing them examples, over and over and over again," says Grasemann. "Every time you show it an example, you say, if this is the input, then this should be your output, and if this is the input, then that should

be your output. You do it again and again thousands of times, and every time it adjusts a little bit more towards doing what you want. In the end, if you do it enough, the network has learned."

In order to model hyperlearning, Grasemann and Miikkulainen ran the system through its paces again, but with one key parameter altered. They simulated an excessive release of dopamine by increasing the system's learning rate—essentially telling it to stop forgetting so much.

"It's an important mechanism to be able to ignore things," says Grasemann. "What we found is that if you crank up the learning rate in DISCERN high enough, it produces language abnormalities that suggest schizophrenia."

After being re-trained with the elevated learning rate, DISCERN began putting itself at the center of fantastical, delusional stories that incorporated elements from other stories it had been told to recall. In one answer, for instance, DISCERN claimed responsibility for a terrorist bombing.

In another instance, DISCERN began showing evidence of "derailment"—replying to requests for a specific memory with a jumble of dissociated sentences, abrupt digressions and constant leaps from the first- to the third-person and back again.

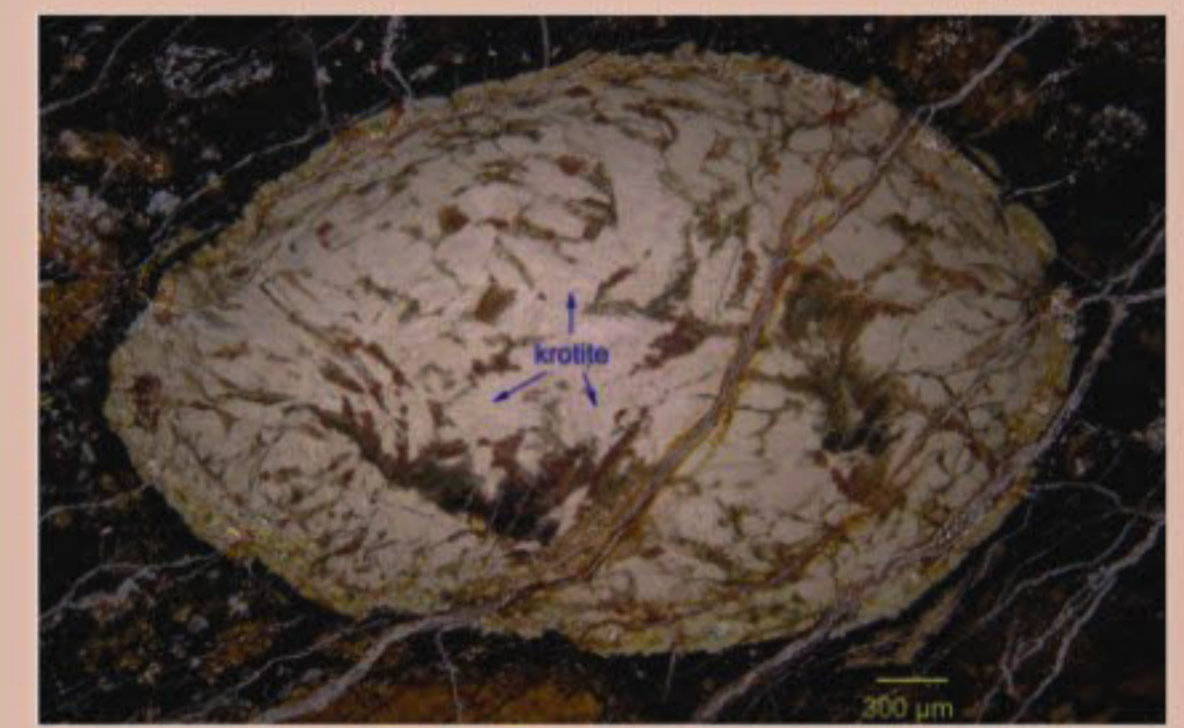
"Information processing in neural networks tends to be like information processing in the human brain in many ways," says Grasemann. "So the hope was that it would also break down in similar ways. And it did."

The parallel between their modified neural network and human schizophrenia isn't absolute proof of the hyperlearning hypothesis is correct, says Grasemann. It is, however, support for the hypothesis, and also evidence of how useful neural networks can be in understanding the human brain.

"We have so much more control over neural networks than we could ever have over human subjects," he says. "The hope is that this kind of modeling will help clinical research."

Source: Science Daily

## Ancient meteorite holds surprise



Krotite mineral in egg-shaped grain (called "cracked egg") in the ancient meteorite

A 4.5-billion-year-old meteorite from northwest Africa has yielded one of the earliest minerals of the solar system.

Officially called krotite, the mineral had never been found in nature before, though it is a man-made constituent of some high-temperature concrete, according to study researcher Anthony Kampf, curator of Mineral Sciences at the Natural History Museum of Los Angeles County (NHM).

"This is one that simply was not known in nature until we found it here," Kampf told LiveScience. "That's pretty dramatic."

The meteorite containing krotite is called NWA 1934 CV3 carbonaceous chondrite. Chondrites are primitive meteorites that scientists think were remnants shed from the original building blocks of planets. Most meteorites found on Earth fit into this group.

The mineral, a compound of calcium, aluminum and oxygen, needs temperatures of 2,732 degrees F (1,500 degrees C) to form, supporting the idea that it was created as the solar nebula condensed and the planets, including Earth, were formed, the researchers say.

The tiny mineral sample just 0.2 inches (4 millimeters) long came from a grain in the meteorite dubbed "cracked egg" for its appearance. In addition to krotite, the cracked egg grain contains at least eight other minerals, one of which is new to science, the researchers say.

Studying this mineral and other components of the ancient meteorite are essential for understanding the origins of the solar system, the scientists say.

Source: Live Science



EVOLUTION VS HEREDITY



LOST BALANCE

## Adventurous gene



Thrill-seeking genes?

AS humans migrated out of Africa around 50,000 years ago and moved across the planet, evolution may have latched onto a gene linked to risk-taking and adventurousness.

The idea, first put forward by Chuansheng Chen at the University of California, Irvine, more than a decade ago, was originally met with scepticism. Now Luke Matthews of Harvard University and Paul Butler of Boston University have shown that a link between two versions of a specific gene and ancient migration patterns stands up to rigorous analysis.

The DRD4 gene codes for a dopamine receptor in the brain. It exists in several versions, or alleles, and studies have shown that people tend to have slightly different personality traits depending on which they have. The 4R allele, for instance, is associated with being even-tempered, reflective and prudent. The less common 7R and 2R versions have been linked to impulsive and exploratory behaviour, risk-taking and the ability to shrug off new situations. Matthews and Butler think that migrants with these versions were better able to deal with dangerous, fluctuating situations and more likely to survive and reproduce under those conditions.

Source: New Scientist

## Unnatural selection: Fish growing up fast

IF we humans are good at hunting, we excel at fishing. As we vacuum up stupendous numbers of fish from oceans, rivers and lakes, the nature of the ones that get away is changing at an astonishing rate.

In particular, the targeting of big animals drives the evolution of smaller fish or ones that become sexually mature at a younger age, or both. Many fish populations are changing dramatically, with average size shrinking by 20 per cent and average life histories 25 per cent shorter (Proceedings of the National Academy of Sciences, vol 106, p 952). Harvested species show the most abrupt trait changes ever observed in wild populations, Michael Kinnison of the University of Maine and colleagues reported recently.

Such changes have been documented in many places, including: pilchard off West Africa; American plaice off Newfoundland; Atlantic salmon in Canada and the UK; herring and grayling off Norway; chum salmon in Japan; sole, haddock and European plaice in the North Sea; whitefish in Alberta; sockeye salmon in Alaska; chum salmon, coho salmon and pink salmon in British Columbia; shellfish in California and cod just about everywhere. What's more, this long list includes only the cases where rigorous studies have been done. It's likely such changes are occurring in every population where large fish are targeted.

So are these changes really the result of evolution, or merely a temporary response to environmental pressures rather than the result of genetic changes? "It is very likely a mixture of



Fishing for a change

the two," says Kinnison. "Evolution is a substantial component."

In several cases, for instance, researchers have shown once fish reach a certain size and age, they are more likely to become sexually mature than previous generations. This rules out most environmental explanations, such as that the fish are maturing at an earlier age because they are finding more food and growing faster.

There is also no doubt about the plausibility of such rapid evolution. A decade-long study of Atlantic silversides kept in tanks has shown that intense targeting of large individuals can halve average size in just four generations.

Actually tracing the genetic changes involved has not been feasible until recently, but it has now been done for Atlantic cod off Iceland. Cod

living with one variant of the Pan I gene live in shallow inshore waters, while those with another variant live in deeper waters further offshore. Because fishing boats target cod in shallow coastal waters, the shallow-living variant is rapidly becoming less common.

Some of the management measures are making matters worse. The deep-living cod do move to shallow coastal areas to spawn, but these areas are closed to fishing during spawning, so the shallow-living fish always bear the brunt of fishing. In the worst case scenario, shallow-living fish could disappear in 15 years. That could lead to the collapse of the fishery, because deep-living cod are much harder and thus costlier to catch.

Source: New Scientist

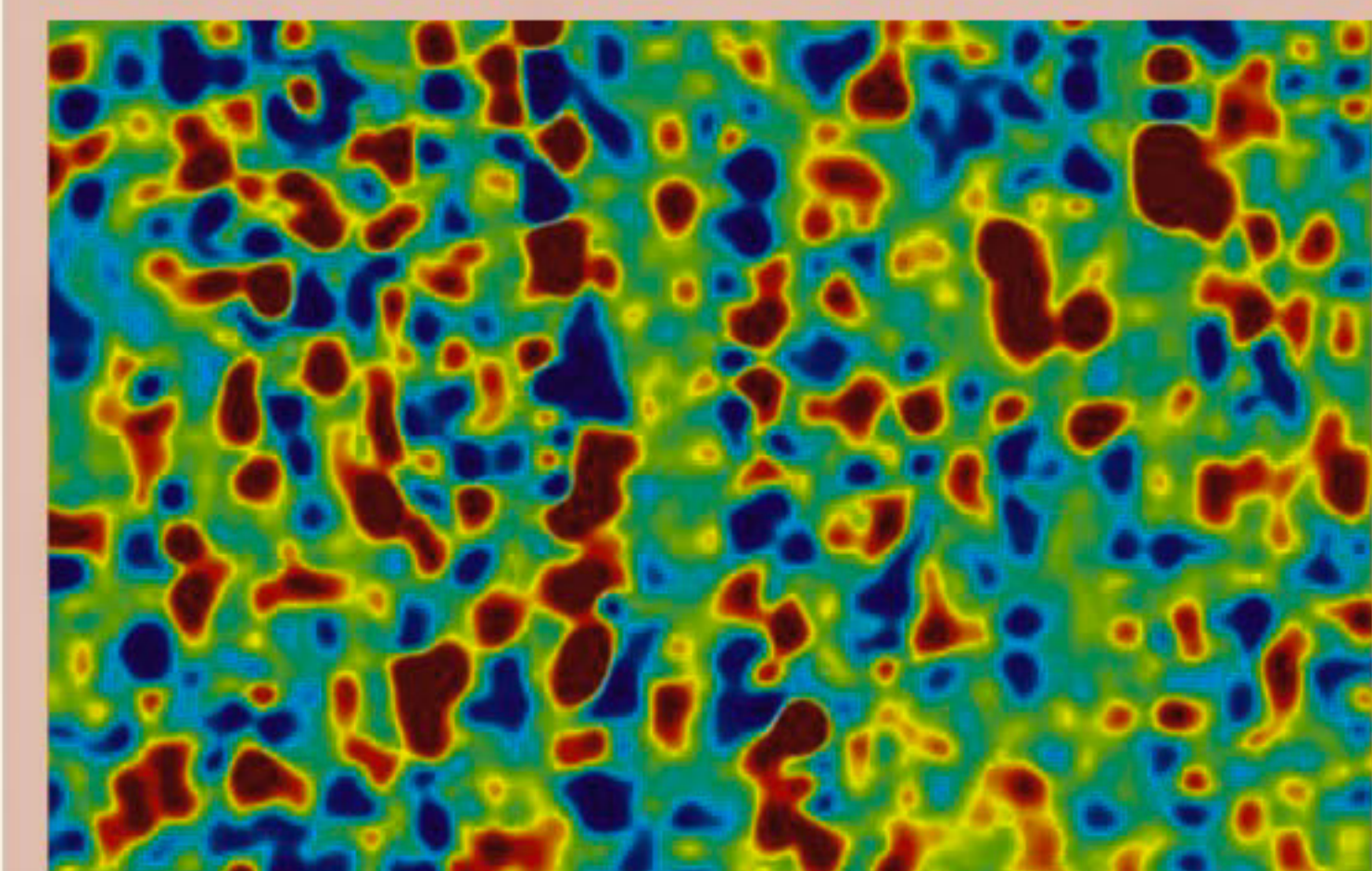


INTO MIST OF TIME



DO YOU KNOW?

## Window to the Ancient Universe



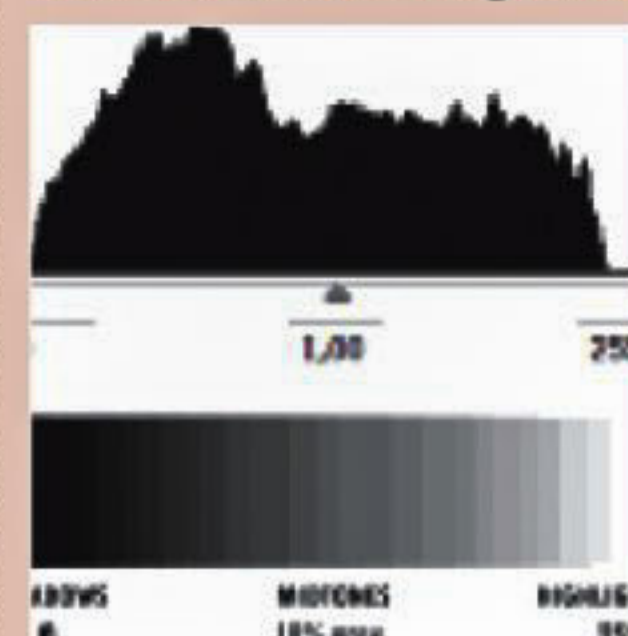
The largest-ever three-dimensional map of the distant universe has been created using the light of the brightest objects in the cosmos.

Since this distant light took eons to reach Earth, the map is essentially a window back in time, providing an unprecedented view of what the universe looked like 11 billion years ago.

"Here, we are looking at intergalactic hydrogen gas, which blocks light," said researcher Anže Slosar, a physicist at the U.S. Department of Energy's Brookhaven National Laboratory. "It's like looking at the moon through clouds -- you can see the shapes of the clouds by the moonlight that they block."

Source: Live Science

## What is a histogram in photography?



Histogram is a very useful tool in photography, found on all the digital cameras (compact cameras and DSLR cameras), as well as in all the photography editing programs. With the histogram you can analyse the tonal value of the image or in other

words: it shows how bright or dark the image is (number of tones captured at each brightness level). The color histogram additionally shows the amount of specific colour in the image



BLESSING IN DISGUISE

## Sickle-cell may blunt malaria

SICKLE-CELL hemoglobin may gas malaria into submission, a new study proposes. People who carry a mutation that deforms the oxygen-carrying protein in red blood cells are well-known to be protected against malaria. Scientists used to think the mutation prevented the malaria parasite from getting into blood cells. But researchers led by Miguel Soares at the Instituto Gulbenkian de Ciência in Oeiras, Portugal, now suggest another mechanism.

The sickle-cell mutation leads to higher production of a protein called heme oxygenase-1, which makes carbon monoxide gas. The gas helps reduce inflammation and protects against death in mice with malaria infection in their brains, the team reports in the April 29 Cell.

Using mice genetically engineered to make human hemoglobin with the sickle-cell mutation, Soares and his colleagues discovered that the mutation primes the body to deal with the red blood cell-shredding activities of the malaria parasite.

Hemoglobin breaks down more easily in people with the sickle mutation, Soares says, releasing a toxic compound called heme. To compensate, the body makes more of the heme oxygenase-1 enzyme, leading to more carbon monoxide production. The carbon monoxide then latches on to hemoglobin and prevents heme from popping off and causing more trouble.

"Before you get infected, sickle hemoglobin is releasing tiny amounts of heme. Your body looks at it and says, 'This could be very dangerous, so I'm going to shut it down,'" Soares says.

Priming the body to deal with the extra heme also means that cells are prepared for the effects of the malaria parasite. The parasite can still infect cells, but the host doesn't get as sick, allowing more time for the immune system to deal with the infection. "It's a politician's approach; you open a dialog with it while you're still fighting it."

Source: Science News



People who carry one copy of a mutation that leads to sickle-cell disease are protected from the worst effects of malaria, research shows. A sickled red blood cell is shown (top) alongside normal red blood cells