RESEARCH HIGHLIGHTS

Weighing a river

Geophys. Res. Lett. **32,** L16308 (2005)
A GPS station in the Amazon basin has registered flex in the Earth's crust caused by seasonal changes in the weight of the great river.

Michael Bevis of Ohio State University in Columbus and his colleagues compared altitude measurements from a ground station of the Global Positioning System near Manaus in Brazil (pictured) with data from an adjacent river gauge. The daily motions of the ground mirrored that of the water level. The height of the station oscillated by 50–75 mm over an annual cycle, while the water level varied by 10–15 metres. The researchers modelled the data as an elastic response of the Earth's crust and upper mantle to flooding within 200 kilometres of the station.

NASA

MOLECULAR DYNAMICS

Fixed on fractals

Phys. Rev. Lett. **95,** 098106 (2005)
An intriguing result in protein dynamics can be explained by the molecules' fractal nature, find Rony Granek of Ben Gurion University and Joseph Klafter of Tel Aviv University, both in Israel.

Recent experimental studies suggested that the relationship between the relative positions of two points on a particular protein — represented by an autocorrelation function — changes in an unusual fashion. As the protein vibrates, this function decays very slowly, first as a stretched exponential and then as a power law.

Granek and Klafter were able to simulate this behaviour using fractal structures to describe the way in which proteins fill space, showing that the phenomenon is not unique to the protein in which it was observed.

NEUROBIOLOGY

Light sensitive

Neuron **47,** 739–750 (2005)
Adding to the already complex picture of how nerve cells operate, a team reports that action potentials, or spikes, can originate in the tufted dendrites of the retina's output neurons.

The dendrites of retinal ganglion cells receive input currents from other retinal cells. These are thought to be combined in the cell's body, or soma, which controls whether a spike is fired down the cell's axon. But after blocking spike production in the soma, researchers led by Rowland Taylor of Oregon Health and Sciences University in Beaverton, unmasked smaller spikes that started in the dendrites in response to light. They suggest the dendritic spikes may underlie the ability of some ganglion cells to detect the direction of motion of a light stimulus.

A. PASIEKA/SPL

CELL BIOLOGY

Err on a G string

Cell **122,** 633–644 (2005)
Spinocerebellar ataxia type 1 is a neurodegenerative disease, characterized by balance and coordination difficulties, that slowly destroys the Purkinje cells (pictured) in the cerebellum.

It is caused by a faulty gene whose protein product, ataxin-1, contains a longer than normal string of glutamine amino acids. This changes the protein's conformation, and also slows its metabolic breakdown.

Huda Zoghbi from Baylor College of Medicine in Houston, Texas, and her colleagues show that the resulting build-up of abnormal ataxin-1 enhances its interactions with Gfi-1. The transcription factor Gfi-1 is essential for the survival of Purkinje cells in adults, and the researchers find that ataxin-1 promotes its destruction.

IMAGE UNAVAILABLE FOR COPYRIGHT REASONS

EPIGENETICS

Mapping yeast

Cell **122,** 517–527 (2005)
Improved high-resolution techniques have produced the first genome-wide map of gene regulation in yeast.

Richard Young and his colleagues at the Whitehead Institute for Biomedical Research in Cambridge, Massachusetts, applied techniques including DNA microarrays containing 40,000 DNA probes to the yeast genome. The resulting map details the location of the histone proteins that organize the structure of DNA. It also shows where the histones have been modified by the addition of chemical groups (that is, by acetylation or methylation), which in turn switches genes on and off.

MEDICINE

Two for one

J. Exp. Med. **202,** 1–10 (2005)
A new vaccine can protect mice against two different fungal pathogens. A team headed by Antonio Cassone of the Italian National Institute of Health in Rome designed the vaccine to sensitize the animals' immune system to a type of large sugar molecule found in many fungal cell walls — β-glucan.

Conjugating the sugar with a carrier protein culled from a diphtheria toxin increased the vaccine's potency. More than 60% of vaccinated mice survived for 30 days following exposure to the fungal pathogen *Candida albicans*, compared with 20% of a control group. And the vaccine nearly tripled the survival rate of mice dosed with *Aspergillus fumigatus* spores.

MOLECULAR BIOLOGY

Immortal coils

J. Cell Biol. **170,** 721–732 (2005)

Since the 1970s, scientists have reasoned that it makes sense for stem cells to keep the original DNA when they divide because it contains the fewest replication errors. Philip Karpowicz of Canada's University of Toronto and his colleagues now offer evidence from mouse neural stem cells to support this debated theory, known as the immortal strand hypothesis.

The team labelled the DNA in a neural stem cell with a compound called BrdU. This cell then divided to form a cluster of cells. The BrdU-labelled DNA appeared in fewer cells than would be expected if the strands from the original chromosomes had dispersed randomly. However, such asymmetric segregation was not seen in other types of stem cells from mice, suggesting the neural stem cells represent a special case.

CANCER

Multitasking

Genes Dev.
doi: 10.1101/gad.1339905 (2005)

The tumour-suppressor gene p53 can be transcribed in six different ways, report researchers led by Jean-Christophe Bourdon and David Lane of the University of Dundee, UK.

The researchers showed that expression of the resulting protein products varies between tissues, and speculate that the interplay between these different protein isoforms is key to p53's normal role, and could even underpin the progression of cancers.

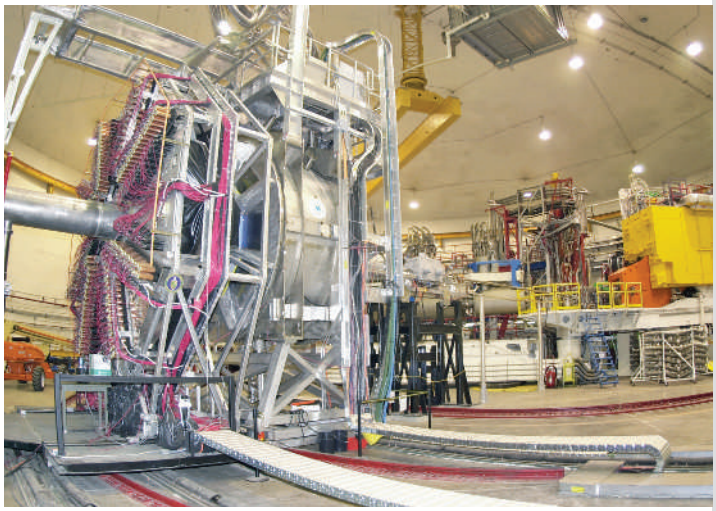
The team found that, like the related genes p63 and p73, the human p53 gene contains an internal promoter region. This means transcription can start in the middle of the gene, with partial transcription giving rise to the short isoforms of the p53 protein. The same internal promoter is conserved across species, indicating its importance. But what regulates the promoter is unclear.

MEDICINE

Blood relations

J. Clin. Invest. doi:10.1172/JCI24177 (2005)

The myelodysplastic/myeloproliferative diseases are a group of disorders in which blood stem cells fail to develop properly — leading to abnormal development of blood cells from bone-marrow precursors. Studying these diseases has been extremely difficult because there has been no single gene associated with them, or animal model of the disorders. Now researchers led by Carlos Martinez-A of the National Center for Biotechnology in Madrid and Miguel Campanero of the Autonomous University of Madrid have tackled both these problems at once. They identify a gene called *Dido* whose altered expression causes myelodysplastic/myeloproliferative-like diseases in mice, and show that altered expression of the gene also occurs in people with the diseases.



JEFFERSON LAB

PARTICLE PHYSICS

Ups and downs

Phys. Rev. Lett. **95,** 092001 (2005)

Inside the proton is a flurry of activity: three permanent quarks, two ups and a down, are surrounded by quark–antiquark pairs that zip in and out of existence. These fleeting 'sea quarks' — primarily strange quarks — contribute to the proton's magnetism and charge.

Now the G0 Collaboration, which is based at the Jefferson Lab in Virginia, Newport News, present a detailed picture of this strange sea. This was created by scattering fast, polarized electrons off protons within a hydrogen target (G0 detector pictured). The data suggest that strange quarks contribute about 5% of the proton's magnetic moment. This is consistent with previous experiments, and highlights a discrepancy with the latest theoretical prediction, which is ten times smaller and has the opposite sign.

JOURNAL CLUB

Jennifer Doudna
University of California, Berkeley

A biochemist exchanges her ideas about molecular motion.

Proteins that convert chemical energy into physical motion fascinate me because they turn the mundane into the extraordinary. They use the simple act of adding water to nucleotides, such as GTP, to drive everything from molecular shape changes to the movement of molecules within cells.

One of their most familiar roles is in helping the ribosome to stitch together amino acids into protein chains. I learnt about this process as an undergraduate, and followed the field through the explosion of interest sparked by high-resolution structures of the ribosomal subunits — so I assumed that I knew the basics.

In the classical model of protein synthesis, the ribosome ratchets along messenger RNA, gathering the amino acids encoded in the RNA sequence. The growing protein chain is extended when the elongation factor EF-G binds to the ribosome, bringing with it a molecule of GTP. Hydrolysis of the GTP drives the addition of the next amino acid by propelling the ribosome along the messenger RNA, which in turn triggers the used EF-G to dissociate. Textbook simple, right?

Perhaps not. A recent paper in the *Journal of Biology* (A. V. Zavialov, V. V. Hauryliuk and M. Ehrenberg **4,** 9; 2005) has made me reconsider this model. It provides evidence that EF-G binds to the ribosome attached to a nucleotide known as GDP, rather than the energy-providing GTP. The ribosome then catalyses the exchange of GDP for GTP, which suggests that it plays a previously unrecognized part in accelerating protein synthesis.

The findings focus new attention on the role of GDP and GTP exchange in driving molecular motion — and show that an established field can still spring surprises.