RESEARCH HIGHLIGHTS

Seafloor snacks

Biol. Lett. doi:10.1098/rsbl.2005.0374

Fossilized sea turtles from the extinct family Protostegidae have been found with bellies full of shellfish. The broken shells in the 110-million-year-old turtles' guts reveal that these ancient sea creatures fed on bottom-dwelling bivalves.

This is surprising, reports Benjamin Kear of the University of Adelaide, who discovered some of the fossils, because protostegids were thought to be surface feeders. Some of their modern relatives, including *Caretta* (pictured), eat mussel-like shellfish from the sea floor. But protostegids had primitive limbs thought to be unsuited to diving. One solution to this puzzle might be that they lived in shallow water. The fossils were found in the Toolebuc Formation in Queensland, Australia.

IMAGE UNAVAILABLE FOR COPYRIGHT REASONS

H. HALL/OSF

GEOPHYSICS

After the earthquake

Geophys. Res. Lett. **32**, L20807 (2005)

The Sumatra earthquake last December not only sent a tsunami rushing across the sea, but also shot sound waves into space. This allowed a remarkable observation to be made — how a natural terrestrial phenomenon affects the magnetic field surrounding Earth.

By comparing data from Thailand, China and Japan, Kyoto University's Toshihiko Iyemori and his colleagues found localized long-wavelength pulsations in the geomagnetic field at the Thai city of Phimai. The timing and nature of the pulsations suggest that the magnetic disturbance was caused by sound waves from the earthquake.

Iyemori has also found that the massive 1991 Pinatubo volcanic eruption in the Philippines affected the magnetosphere.

DRUG DISCOVERY

Immune booster

Nature Chem. Bio. doi:10.1038/nchembio746 (2005)

By combining knowledge of chemical structure with an understanding of biological function, researchers have designed simple molecules that bind to a tumour necrosis factor (TNF) receptor. Such receptors help to control immune responses, so TNF impostor molecules could form the basis of therapies to boost or suppress the immune system.

Sylvie Fournel and Gilles Guichard, both at the Institute of Cellular and Molecular Biology in Strasbourg, France, and their colleagues made the small molecules by synthesizing the parts of a protein known as CD40L that bind it to its TNF receptor. They attached these protein parts to a central scaffold that had the same threefold symmetry as the protein's receptor.

MICROBIOLOGY

Cells come unstuck

J. Exp. Med. **202**, 1-13 (2005)

In vitro experiments are unravelling details of how the bacterium *Helicobacter pylori* (pictured) causes gastric ulcers, the discovery that won this year's medicine Nobel Prize.

A protein produced by *H. pylori*, called CagA, is known to disrupt the protective lining of the stomach. Researchers led by Chihiro Sasakawa, of the University of Tokyo, show that it does this by binding to Crk proteins in the host cells. The hijacking of the Crk proteins activates signalling pathways that control cell turnover. This ultimately leads to the loss of the adhesion molecules that help the cells of the stomach lining to stick together, causing cell scattering.

IMAGE UNAVAILABLE FOR COPYRIGHT REASONS

CELL BIOLOGY

Waste not, want not

Cell **123**, 423-436 (2005)

The cell's waste-disposal machine, the proteasome, has hidden talents. As well as degrading unwanted proteins, this enzyme has a key role in controlling the activity of certain genes, say US researchers.

A team led by William Tansey, of the Cold Spring Harbor Laboratory in New York, and Jerry Workman, at the Stowers Institute for Medical Research in Kansas City, Missouri, has studied in yeast one of the two main parts that make up this complex enzyme. They found that it draws another collection of proteins — known as the SAGA coactivator complex — to the control regions of certain genes. This complex alters the chemistry of the proteins that package DNA, so altering gene activity.

STRUCTURAL BIOLOGY

Engines of creation

Science **310**, 827-834 (2005)

Of all the celebrated molecular machinery in biology, the most vital is surely the ribosome. This structure turns the genetic data of RNA into proteins and nucleic acids.

But the ribosome is complex, which is why the atomic-resolution crystal structure of the ribosomes in the bacterium *Escherichia coli* has been so long in coming. Jamie Doudna Cate, of the University of California, Berkeley, and his co-workers at last show us what the ribosomes of this archetypal prokaryote look like. The unprecedented level of detail gives a clearer picture of how the intricate protein factory functions.

SPL

CANCER

Catch it quick

Proc. Natl Acad. Sci. USA **102,** 16368–16373 (2005)

Hopes of detecting cancers early and non-invasively by searching for mutant DNA in patients' blood plasma have been put to the test in a study of people with colorectal cancer.

The results, collected from 33 patients with colorectal tumours and 10 without, indicate that even low levels of mutant DNA from cancerous cells can be picked up. The technique identified malignant tumours, often before they spread, but not premalignant masses. The plasma DNA was analysed using a sequencing assay developed for the purpose, report researchers led by Bert Vogelstein of the Johns Hopkins Medical Institutions in Baltimore, Maryland. They propose that the abnormal DNA is released into the blood when tumour cells are engulfed by immune cells.

BIOTECHNOLOGY

Tiny tweezers

Lab Chip **5,** 1224–1228 (2005)

Living *Escherichia coli* bacteria can be teased into a three-dimensional arrangement inside a gelatin sample using optical tweezers, researchers have shown. The technique, invented by a team of scientists from Sweden and Britain, could be used to study cell function or to build templates for growing replacement tissue.

Tweezers made from infrared laser beams were used to move the bacteria around in the liquid gelatin before it set. Once the cells were fixed in place, the lasers were switched off. Provided with the appropriate nutrients, the bacteria could survive within the gelatin matrix for several days.

PHYSICS

Spins in sync

Nature Phys. **1,** 111–116 (2005)

Experiments have confirmed that the quantum characteristics of a Bose–Einstein condensate extend to the spin of its atoms.

Michael Chapman and colleagues from the Georgia Institute of Technology, Atlanta, made their quantum gas condensate by cooling rubidium atoms until they all dropped into the same quantum state. Using magnetic fields, the researchers were able to visualize the condensate's spin, which can point in one of three directions. They also saw that interactions between condensates with different spins were reversible, unlike spin interactions in a classical gas.

IMAGE UNAVAILABLE FOR COPYRIGHT REASONS

M. THOMAS/SPL

CELL BIOLOGY

Tipped for protection

Genes Dev. doi:10.1101/gad1293805 (2005)

Telomeres, which cap the tips of chromosomes, inhibit the signalling triggered by double-stranded breaks in DNA, report Ted Weinert, of the University of Arizona in Tucson, and his colleagues. This explains how telomeres help to prevent the cell's DNA-repair machinery from fusing chromosome ends.

Weinert's group compared the response of yeast cells to a double-stranded break near to a telomere sequence with a break elsewhere. The telomere sequences seem to act as 'anti-checkpoints', suppressing the recruitment of the checkpoint proteins that normally suspend the cell cycle while the damage is repaired.

IMMUNOLOGY

The enemy within

Science **310,** 850–855 (2005)

A deeper understanding of how the immune system avoids attacking our own cells may point the way towards a therapy for the autoimmune disease multiple sclerosis.

The amino acid tryptophan has previously been associated with control of 'anti-self' immune reactions. Lawrence Steinman of Stanford University, California, and his colleagues found that its action is triggered by the enzyme indoleamine 2,3-dioxygenase. Cells carrying 'self' markers boost transcription of this enzyme, which breaks down tryptophan. In a mouse model of multiple sclerosis, the products of the tryptophan breakdown, and similar molecules, shut down anti-self reactions and eased symptoms of the disease.

JOURNAL CLUB

Steven Salzberg
University of Maryland, College Park, USA

A genomics researcher wonders why viruses don't get the respect they deserve.

After sequencing hundreds of flu genomes this year (E. Ghedin *et al. Nature* **437,** 1162–1166; 2005), my colleagues and I constantly find ourselves discussing the flu virus as if it were a living organism. We try to be careful to say 'particle' instead of 'cell', and 'segment' instead of 'chromosome'. But are we right to imply that viruses are alive?

Like many students, I learned in high school that viruses were not alive. And I never had cause to question this until last year, when Jean-Michel Claverie visited my group to talk about the sequencing of the 'giant' virus known as mimivirus.

Claverie and his colleagues had just claimed in *Science* (D. Raoult *et al.* **306,** 1344–1350; 2004) that this organism has so many capabilities it deserves its own branch on the tree of life. The evidence is compelling: mimivirus has a double-stranded DNA genome of 1.2 million base pairs, with many genes never before seen in viruses, including several necessary for protein synthesis and even DNA repair.

Not surprisingly, this study provoked controversy. Some argued (D. Moreira & P. López-García *Science* **308,** 1114; 2005) that mimivirus is just an unusually adept 'gene pickpocket', scavenging genes from its hosts.

Claverie's team offered a spirited defence, saying the genes' ancient origin supports the idea that viruses have gradually trimmed down since they originated near the base of the tree of life, shedding genes to rely increasingly on their hosts.

Viruses have survived for millions of years, evolving and adapting just like 'real' life forms. But if we accept that they branched from the tree of life, we will face another dilemma: how much original genetic material must they have lost for us to stop calling them life? The tiny influenza virus, with just 11 genes, is waiting to see if we can figure it out.