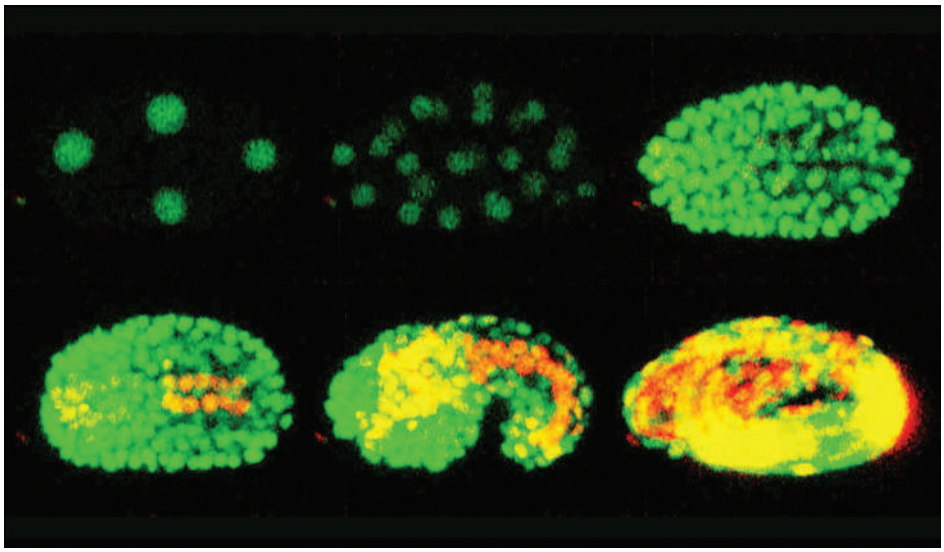
RESEARCH HIGHLIGHTS

Follow that cell

Proc. Natl Acad. Sci. USA **103,** 2707–2712 (2006)
A cell-tracking system developed in living worm embryos could help to reveal the function of developmental genes.

Robert Waterston of the University of Washington in Seattle and his colleagues labelled the nuclei of *Caenorhabditis elegans* embryonic cells with green fluorescent protein and snapped 30 microscope images (examples pictured) every minute for eight hours as the embryo grew. The team built an algorithm that can figure out from these images which cell gave rise to which others. The algorithm takes just 25 minutes to run on a desktop computer.

The system can be used in combination with probes that monitor gene activity (appearing red in images).



A. WATERSTON, Z. BAO & J. MURRAY

CHEMISTRY

Fantastic plastic

Angew. Chem. Int. Edn doi:10.1002/anie.200504241 (2006)
Hydrogen cars need a safe and efficient way of storing the combustible gas. Could polymers be the answer?

Organic polymers are generally too floppy to form rigid, porous structures. But a recently reported class of organic polymer has molecules that contain bulky segments, opening up spaces in their packing. Neil McKeown of Cardiff University, UK, Peter Budd of the University of Manchester, UK, and their colleagues have now shown that such polymers can trap up to 1.7% of their own mass in hydrogen at − 196 °C.

That's still a long way from the US Department of Energy's goal to have a system that can store 6% hydrogen (by mass) by 2010. But organic polymers have great potential for structural fine-tuning, the team argues.

IMMUNOLOGY

One step to gene set

Nature Methods doi:10.1038/nmeth858 (2006)
A new kind of transgenic mouse should speed up the study of T cells, which help the immune system to tell 'self' bodies from non-self invaders.

Creating transgenic mice with different combinations of T-cell receptors can be a time-consuming endeavour, typically involving many rounds of cross-breeding.

Now Dario Vignali and his colleagues at St. Jude Children's Research Hospital in Memphis, Tennessee, have acheived this in just one step.

They used specialized retrovirus vectors to insert various T-cell receptor genes into mouse bone-marrow stem cells. These cells were then transplanted into T-cell-deficient mice, which started producing T cells with the desired receptors in five to eight weeks. Traditional methods would have taken six months.

GEOLOGY

Under ice

Geophys. Res. Lett. **33,** L02504 (2006)
Lake Vostok — a vast, ancient lake buried beneath the Antarctic ice sheet — has been found to have two similar neighbours (pictured below). Such lakes might harbour exotic forms of life.

The two lakes have areas of around 1,600 and 2,000 square kilometres. They seem, like Lake Vostok, to be controlled by local tectonics. This could mean that the lakes have been stable for hundreds of thousands of years.

To make the find, Robin Bell of Lamont-Doherty Earth Observatory in Palisades, New York, and her team analysed satellite altimetry data, aircraft-borne gravity measurements and ground traverse data.

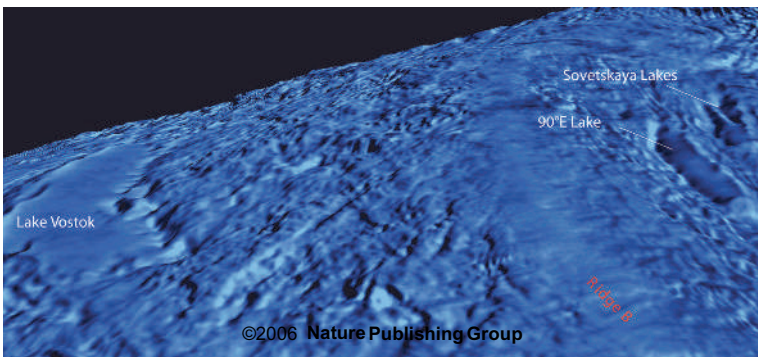
GENE THERAPY

Haemophilia on hold

Nature Med. doi:10.1038/nm1358 (2006)
Medical researchers have used gene therapy to provide a temporary cure for severe haemophilia B.

In a clinical trial of seven patients, Katherine High of the Children's Hospital of Philadelphia, Pennsylvania, and her team injected sufferers with a modified virus carrying the gene for blood coagulation factor IX (F.IX), which is mutated in haemophilia B patients. The gene was incorporated into liver cells and enough F.IX was produced to alleviate the uncontrolled bleeding that characterizes the condition.

The effect lasted about eight weeks, after which the immune system is thought to have banished the modified liver cells. Using a different viral vector or administering immunosuppressing drugs may provide more permanent treatment, the researchers hope.



C. SHUMAN & V. SUCHDEO/NASA GODDARD CENTER; M. FAHNESTOCK/UNIV. NEW HAMPSHIRE

VIROLOGY

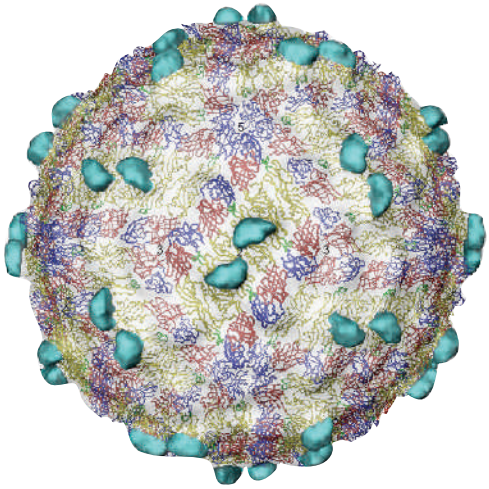
Fearful asymmetry

Cell **124,** 485–493 (2006)
A team of researchers in the United States has revealed how the unusual outer structure of the dengue virus

(pictured right) helps it to infect cells.

The hollow protein shells of viruses are made of protein units packed together like the panels on a soccer ball. In most viruses, each unit consists of a group of identical proteins arranged symmetrically. But the three proteins in the dengue virus unit turn out to be arranged asymmetrically.

Michael Rossmann of Purdue University in West Lafayette, Indiana, and his colleagues worked out how these proteins bind to a cell-surface protein known as DC-SIGN. Thanks to the asymmetry, DC-SIGN binds to sugar molecules on just two of the proteins, sticking the virus to the cell in a way that aids its invasion.



CANCER

Why the drugs don't work

Cell **124,** 615–629 (2006)

Prostate cancer responds at first to drugs that block the action of androgens, such as testosterone, on which its growth depends. But resistance to the drugs soon develops.

Now Michael Rosenfeld and David Rose of the University of California, San Diego, and their colleagues report an unexpected mechanism that may explain why the drugs stop working.

They traced the effect to a chemical produced by macrophages — immune cells that infiltrate prostate tumours. The chemical produced, a cytokine, triggers a chain of reactions that ultimately reactivate the blocked androgen receptor.

The effect is mediated by a particular domain of the receptor. The same domain is found in all sex steroid receptors, and may have evolved for fine-tuning of hormone activity during sensitive events in mammalian reproduction.

QUANTUM PHYSICS

Clone rangers

Phys. Rev. Lett. **96,** 060504 (2006)

A quantum scheme dubbed 'telecloning' has been realized by researchers at the University of Tokyo in Japan, and the University of York in the United Kingdom.

Telecloning allows a quantum state to be simultaneously copied and reproduced in a number of remote locations. The protocol was implemented using three laser beams, entangled so that their quantum states were linked. First, a measurement was performed on one beam, the 'sender'. The result of that measurement was then used to guide manipulations of the two 'receiver' beams such that both adopted approximations of the quantum state specified by the sender. Quantum mechanics forbids the creation of perfect copies, but the experiments achieved copies almost as good as theory permits.

CHEMISTRY

At knife point

J. Am. Chem. Soc. doi:10.1021/ja057029t (2006)

Simulations suggest that antimicrobial peptides slice through the lipid bilayer of microbial cell membranes like a knife through butter. Now chemists at the University of Michigan in Ann Arbor and the University of Massachusetts in Amherst, led by Gregory Tew and Zhan Chen, have demonstrated a way to watch the blade in action. The technique could help in screening new candidate antibiotics.

The researchers used sum-frequency generation vibrational spectroscopy to follow, in real time, the interaction of a typical antimicrobial peptide and a synthetic bilayer.

The team's observations of the lipid layer's destruction square with previous experiments, which have shown that the peptide inhibits bacteria when its concentration exceeds 0.8 $\mu g\,ml^{-1}$.

DRUG DISCOVERY

Treating progeria

Science doi:10.1126/science.1124875 (2006)

A drug that interferes with a building block of the cell's nucleus could be used to treat Hutchinson–Gilford progeria syndrome (HGPS), a rare premature-ageing condition in which children rarely survive beyond their teens.

Researchers discovered in 2003 that HGPS is caused by a mutant form of lamin A, a key protein in the support scaffold of the nucleus. The mutant protein accumulates at the nuclear rim because a particular lipid section of the molecule behaves abnormally.

Loren Fong of the University of California, Los Angeles, and his colleagues show that drugs known as farnesyltransferase inhibitors, which block the addition of this lipid and are already in clinical trials as cancer drugs, reverse some of the symptoms in a mouse model of the disease.

JOURNAL CLUB

Michelle Peckham
University of Leeds, UK

A cell biologist turns detective to unravel the workings of one of the body's molecular machines.

I'm fascinated by the structure and function of myosins, a large, varied family of motor proteins. These molecules specialize in particular jobs — such as driving muscle contraction — and they move by walking along tracks made of actin.

But understanding how myosins work has been like following a detective story, complete with false leads and unexpected twists.

One important characteristic of a myosin is whether or not it dimerizes; that is, pairs up with another myosin molecule. Dimers can use their two motor domains to walk along the actin, whereas monomers can take just a single step. Processive walking is useful for myosins carrying cargo long distances — such as the class that transports pigments in the skin.

Myosins can be zipped together into dimers by the α-helices in their tails wrapping around each other to form a structure known as a coiled coil. Unfortunately, the computer programs used to predict the formation of a coiled coil from sequence data are not infallible.

For example, for certain subsets of myosins, the programs predict that coiled coils should form, but in reality they do not. This is the case for myosin 6, which, contrary to predictions, has been observed (in some experiments) as a monomer.

So a recent paper showing that myosin 6 can dimerize after all comes as a surprise (H. Park *et al. Mol. Cell.* **21,** 331–336; 2006). The myosins pair up when densely packed on to actin, seemingly without a coiled-coil region, and do so more readily if the tip of the myosin's tail is missing. This hints that cargo binding to the tail could initiate the process.

But will the mechanism operate *in vivo*? And can other myosins dimerize in this way? Or is this just another false lead? I await the next chapter in the story.