

RESEARCH HIGHLIGHTS

Culture shock

Nature Med. doi:10.1038/nm1268 (2005); *Proc. Natl Acad. Sci. USA* doi:10.1073/pnas.0503596102 (2005); *Science* doi:10.1126/science.1114016 (2005)

More than 170 million people worldwide are infected with the virus that causes hepatitis C, a major liver disease. Understanding the life cycle of the virus (pictured) is essential for developing effective treatments, but progress has been limited because the virus is difficult to grow in culture and therefore hard to study.

In independent papers, three teams now report successful *in vitro* systems for propagation, using a unique hepatitis C virus clone derived from a Japanese patient by Takaji Wakita at the Tokyo Metropolitan Institute for Neuroscience. His group, and teams headed by Francis Chisari at the Scripps Research Institute, California, and Charles Rice at Rockefeller University, New York, propagated this clone in a human liver-cancer cell line. Their systems produce high yields of virus that can be used to infect further cells.

IMAGE
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MATERIALS**Foul play**

J. Am. Chem. Soc. **127**, 7972–7973 (2005)

Mussels stick tenaciously to the hulls of boats, and this increases the boats' drag. But Phillip Messersmith and colleagues at Northwestern University in Illinois, Evanston, plan to turn one of their own proteins against them.

They have developed an antifouling polymer that prevents biological adhesion — a non-stick compound is anchored to a surface by a peptide that mimics the adhesive protein of blue mussels (*Mytilus edulis*, pictured). The non-stick element is a polymer made from an artificial analogue of glycine.

The material could have applications not only in marine engineering but also in medicine, for example, to keep implanted

devices clean. Titanium coated with the antifouling polymer remains relatively cell-free in a culture of tissue-forming fibroblasts for months.

IMMUNOLOGY**Poxy antibodies**

Nature Med. doi:10.1038/nm1261 (2005)

More than 200 years after Edward Jenner realized that infection with cowpox made milkmaids resistant to the smallpox virus, a group led by Genoveffa Franchini from the National Cancer Institute in Bethesda, Maryland, has discovered how a smallpox vaccine based on the *vaccinia* virus confers immunity.

Using macaques infected with monkeypox virus, which is a good model for smallpox infection in humans, the researchers showed that the protective power of the vaccine is mediated by the immune system's B cells, rather than its T cells. The B cells produce antibodies that bind specific poxvirus proteins — and the researchers found that antibodies from vaccinated humans protected macaques from severe disease. The finding may assist the search for a safer alternative to the current live-virus vaccine.

NANOTECHNOLOGY**Going for gold**

J. Am. Chem. Soc. doi:10.1021/ja042621o (2005)

Spherical cages of carbon atoms that attach gently to gold should allow more complex molecular patterning of gold surfaces,

according to Paul Weiss's group at the Pennsylvania State University, University Park.

His team persuaded 1-Adamantanethiol molecules to self-assemble into a monolayer on gold. Because these molecules' interactions are weak, they can be displaced by other molecules that bind to gold, such as alkanethiols. This should make it possible to print patterns of molecules, perhaps with conducting or sensing properties, into the 1-Adamantanethiolate layer. The surrounding layer would prevent the pattern spreading by diffusion, overcoming a problem encountered when some molecules are printed on bare gold surfaces.

MEDICINE**Barrier grief**

J. Clin. Invest. **115**, 1607–1615 (2005)

To cause meningitis, *Streptococcus pneumoniae* must find its way across the blood-brain barrier. A group led by Jörg Weber of the Charité Medical School in Berlin, Germany, has now worked out how the bacterium damages the endothelial cells that make up the barrier. They find that the pathogen induces programmed cell death through two different mechanisms. One pathway is triggered by toxins produced by living *S. pneumoniae*, the other by components of its cell wall. The latter mechanism has implications for therapeutic treatment — antibiotics that target the *S. pneumoniae* cell wall might cause further tissue damage through the release of cell-wall debris.

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CELL BIOLOGY

Calling tails

J. Cell Biol. doi:10.1083/jcb.200411001 (2005)

Eggs attract sperm by releasing chemicals that boost calcium ion (Ca^{2+}) levels inside a sperm, so altering the direction in which it swims. But rather than responding to overall levels of Ca^{2+} , as previously thought, the sperm react to the rate at which its concentration changes, reports a team led by Christopher Wood at the National Autonomous University of Mexico, Cuernavaca.

Experiments in sperm from the sea urchin *Arbacia punctulata* suggest that attractant chemicals trigger two waves of Ca^{2+} that pass through the sperm's tail. The first is short and rapid, the second long and slow. Blocking the first rapid flux stops the sperm changing direction even though the second wave elevates Ca^{2+} levels, revealing a surprising complexity in calcium-ion signalling.

QUANTUM PHYSICS

Lamb chops

Phys. Rev. Lett. **94**, 223001 (2005)

The field theory of electromagnetism — known as quantum electrodynamics — is the best-tested theory in physics. But researchers will not rest until they have tested its validity in extremely strong electric fields, such as those generated by heavy nuclei.

To do this, Alexandre Gumberidze of the Heavy Ion Research Centre in Darmstadt, Germany, and his colleagues measured a tiny split in the energy levels of a uranium ion from which all but one electron had been stripped. This made it possible to see the Lamb shift, a split in energy levels usually visible only in the single-electron hydrogen atom. The test was three times more precise than the previous best measurement.

IMAGE
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VIRAL TRANSPORT

Stowaways

Nature Cell Biol. doi:10.1038/ncb1269 (2005)

Certain viruses improve their ability to infect cells by stowing away in structures called internal vesicles, according to a team led by Jean Gruenberg at the University of Geneva, Switzerland. This means that infection takes place in two steps — not one, as previously thought.

The team tracked the vesicular stomatitis virus as it infected cultured cells. The cell swallowed the virus into a structure called an early endosome at the cell's edge, as expected. But instead of escaping straight from the endosome into the outer regions of the cell, the virus within its endosome entered a small bubble-like vesicle. These vesicles only fuse with the outer membrane of the endosome when the complex is deep inside the cell, releasing the virus right next to the cell's nucleus.

WEST NILE DISEASE

Blood wedding

Proc. Natl. Acad. Sci. USA

doi:10.1073/pnas.0503835102 (2005)

The discovery that one mosquito can transmit the West Nile virus directly to another may help explain the surprisingly rapid spread of the disease through North America.

Usually mosquitoes (*Culex pipiens quinquefasciatus*) pick up the West Nile virus by feeding on birds infected with it. But researchers led by Stephen Higgs of the University of Texas Medical Branch in Galveston have shown that the virus can pass between two mosquitoes if they sip blood simultaneously from an uninfected host. This type of transmission has previously been demonstrated in ticks and blackflies. In this case, the recipient mosquito may acquire the virus by directly ingesting infected saliva from a feeding neighbour, but this remains to be proven.

DATA STORAGE

Pillar talk

J. Appl. Phys. **97**, 103910 (2005)

One way to pack more data on to magnetic disks is to pattern the surface, defining small dots that store single bits. Another approach is to use multiple magnetic layers, so more than one bit can be stored per spot.

Combining the two tactics, researchers from the University of Konstanz in Germany and the Hitachi San Jose Research Center in California deposited multiple layers of cobalt and palladium onto a field of silicon pillars, spaced 300 nanometres apart. They stored two bits per pillar, giving higher data densities than otherwise possible with this scale of patterning.

JOURNAL CLUB

John Brookfield
University of Nottingham, UK

A population geneticist ponders the evolution of his field.

Since I started in the business of population genetics, the field has been transformed by the extraordinary increase in size of the data sets. A recent paper in *Science* (D. A. Hinds *et al.* **307**, 1072–1079; 2005) is a prime example of such a set, but we have yet to exploit them.

In the 1970s, studies followed a common pattern. First they

identified a gene with different variants in a population. That meant looking for detectable differences between individuals — for example, as in my PhD, differences in the charges of enzymes. Once a variation in a gene, known as a polymorphism, was identified, they tried to establish whether natural selection was operating on it.

Now that we have DNA sequences, we can find single nucleotide polymorphisms, in which a single letter of the

sequence varies. The authors of the *Science* paper use DNA chips to find the frequencies of more than 1.5 million single nucleotide polymorphisms in three human populations. But how can we use such a wealth of figures to find out about natural selection?

One tantalizing idea is that we can measure — in an objective way — how quickly species are evolving. Genetic differences in a population accumulate randomly over time, so regions of the genome where polymorphisms are rare

must have been swept clean recently by a spreading variant of a gene. These sweeps are evidence of adaptive change.

Counting the number of sweeps that have affected the human genome will, I suspect, tell us that our species is changing rapidly through adaptation. That could explain why we seem so different from our ape relatives.

We might also identify the genes in which these changes have occurred — a perennial goal of human evolutionary genetics.