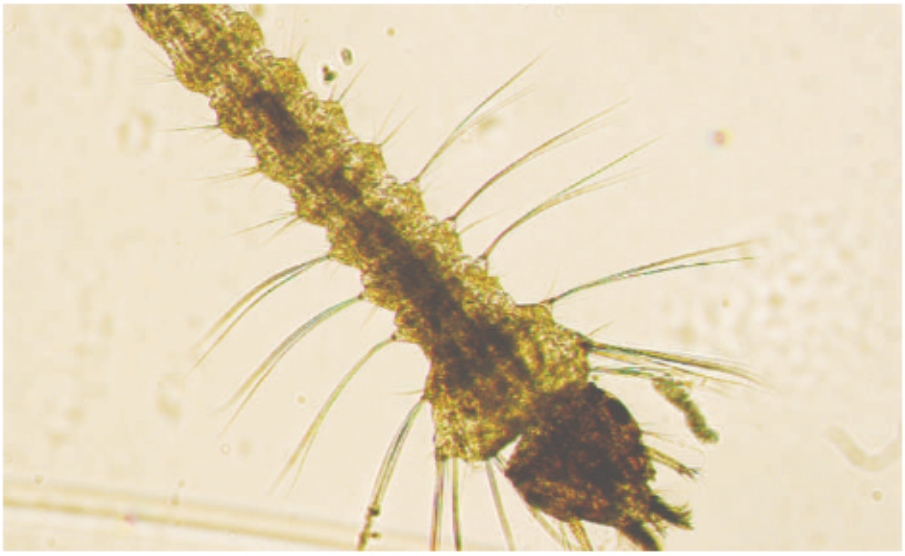
RESEARCH HIGHLIGHTS

Glimmer boys

Nature Biotechnol. doi:10.1038/nbt1152 (2005)

Biologists have genetically engineered mosquitoes to produce males with sperm that glows. The point? The fluorescent gonads make it possible to separate males from females at an early stage of larval development.

Insect control strategies often depend on the release of sterile insects. But in mosquitoes, females are the transmitters of malaria, so it is important to release only males. The glowing genitalia offer a way to isolate a male-only population that can then be sterilized and let out into the wild. The researchers at Imperial College London, led by Andrea Crisanti, carried out their experiments on the mosquito *Anopheles stephensi* (larva pictured right), which is the principal vector of human malaria in Asia.



F. CATTERUCCIA & A. CRISANTI

ZEPHYR/SPL

CELL BIOLOGY

Lipids link to Alzheimer's

Nature Cell Biol. doi:10.1038/ncb1313 (2005)

Elevated levels of the amyloid beta peptide (Aβ) are known to lead to the formation of brain plaques (pictured below, damaged areas in brown) in Alzheimer's disease. But the normal function of Aβ has been a mystery. Now a study reveals a role for it in regulating lipid metabolism.

Tobias Hartmann of the Centre for Molecular Biology in Heidelberg, Germany, and his colleagues engineered cells from mice to lack the machinery that makes Aβ. These cells ended up containing large amounts of cholesterol and lipids.

The researchers also identify Aβ's targets: two enzymes that are both involved in cholesterol and lipid-processing. The work should help to explain the association that has been noticed between Alzheimer's and high cholesterol levels.

IMAGE UNAVAILABLE FOR COPYRIGHT REASONS

MOLECULAR ELECTRONICS

Rattling chains

Phys. Rev. B 72, 121405(R) (2005)

The way molecular vibrations affect the flow of electrons through a chain of atoms has been captured in a new charge transport model.

Developed by researchers from Ohio University in the United States and from the Pontifical Catholic University in Rio de Janeiro, Brazil, the model takes into account the repulsive effects between electrons, and how the molecule's vibrations, called phonons, affect the energy levels that the electrons can occupy. This makes it more realistic than previous models, which focused on only one of these two interactions.

The researchers found that vibrations can open unexpected transport channels, which allow electrons to hop between atoms, or they can block the electrons' flow. These observations may help in modelling the behaviour of electrical components made from single molecules.

ANTIBIOTIC RESISTANCE

Short enzyme stymies drug

J. Biol. Chem. doi:10.1074/jbc.M505727200 (2005)

The troublesome resistance of members of the *Mycobacterium tuberculosis* complex to a new class of macrolide antibiotics called ketolides stems from a truncated form of a gene called *erm*.

Like other *erm* genes, which are found in a diverse range of pathogenic bacteria, the *erm*(37) homologue in *M. tuberculosis* encodes a methlytransferase enzyme with a highly targeted action. By adding a methyl group to a particular nucleotide in a specific ribosomal RNA sequence, this enzyme confers resistance to older macrolide antibiotics, such as erythromycin, but not to the newer ketolides.

Researchers led by Stephen Douthwaite of the University of Southern Denmark, Odense, now show that ketolide resistance arises from the imprecise action of *erm*(37)'s enzyme. The enzyme encoded by the truncated gene slips along its RNA target, adding extra methyl groups to neighbouring nucleotides.

IMMUNOLOGY

Neurons enter the fray

J. Virol. 79, 12893-12904 (2005)

Human neurons are capable of sensing and responding to viral infection, finds a group led by Monique Lafon at the Pasteur Institute in Paris.

Previously, it was thought that neurons relied on infection being detected by their companion glial cells. But Lafon's group discovered that infecting neurons with rabies virus; a herpes virus; or with double-stranded RNA, a molecular signature of RNA viruses, switched on genes involved in immunity.

Neurons were also found to express the protein Toll-like receptor 3, which recognizes and responds to double-stranded RNA. Double-stranded RNA could therefore be the trigger that turns on the innate immune response in human neurons.

CELL BIOLOGY

Comfortably numb

J. Neurosci. **25**, 8924–8937 (2005)

Camphor, the pungent active ingredient of mothballs, produces a warming sensation and mild local anaesthesia when applied to the skin. But although camphor has been used therapeutically for centuries, its mode of action has only just been uncovered.

Using rat neurons, David Clapham and his colleagues at the Howard Hughes Medical Institute in Boston showed that camphor works in the same way as capsaicin. This is the 'hot' compound in chilli peppers, and also a mild analgesic. Both substances activate, and subsequently numb, the heat-sensitive TRPV1 receptor of sensory neurons. Camphor was also found to inhibit the cold-sensitive TRPA1 receptor.

MATERIAL SCIENCE

Bonds writ large

Appl. Phys. Lett. **87**, 131903 (2005)

Even big cracks start small: the collapse of a bridge begins with the breaking of atomic bonds at the tip of a flaw. But studying real materials at an atomic scale during fracture is very challenging. So Francisco Emmerich of the Federal University of Espirito Santo in Brazil is offering an alternative. He has designed a scaled-up solid, using bar magnets stacked in a brick-wall arrangement and separated by layers of foam.

The model solid closely mimics the forces between atoms. Experiments using it show that catastrophic failure always starts in the same way: two of the magnets at the crack tip jump apart to a critical separation. This may be equivalent to a chemical bond breaking at the atomic scale.

EVOLUTIONARY GENETICS

Copy and save

Genome Res. **15**, 1421–1430 (2005)

A key question in genetics is how extra copies of genes manage to persist in an organism. When a gene becomes duplicated, one copy often gets deleted or inactivated. But sometimes both remain intact.

To find out more, Uwe Sauer and his team at the Swiss Federal Institute of Technology in Zurich used computer modelling and quantitative biochemical experiments to study 105 families of duplicated genes in the yeast *Saccharomyces cerevisiae*.

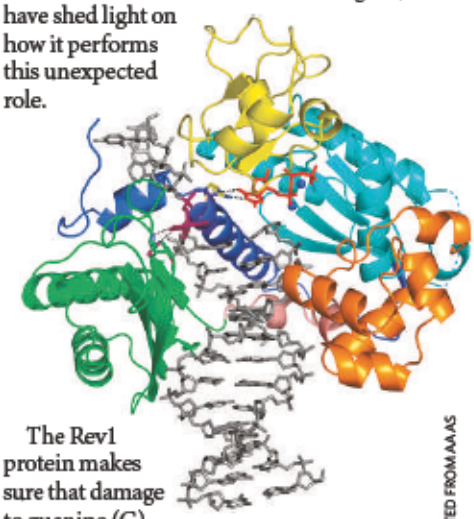
They found that spares adopt four survival strategies: a copy can act as a backup, develop a new function, become regulated differently or simply boost the levels of the protein encoded by that gene. All mechanisms seem to be equally important.

PROTEIN STRUCTURE

Skipping the G spot

Science **309**, 2219–2222 (2005)

The protein pictured below has a remarkable ability: it can stand in for a genetic base when DNA is being copied. By deciphering the crystal structure of the protein, Aneel Aggarwal of the Mount Sinai School of Medicine in New York and his colleagues, have shed light on how it performs this unexpected role.



ADAPTED FROM AAAS

The Rev1 protein makes sure that damage to guanine (G) bases in a DNA template can be bypassed. Without the protein, a spoilt base would block gene replication. Aggarwal's structure shows yeast Rev1 bound to a template G (violet) in a DNA helix (grey). The protein evicts the base from the helix and attracts the G's partner, a cytosine (C) base, to bind to itself. This means the C, carried by the molecule dCTP (red), gets incorporated into the new DNA strand in the right place.

CANCER

Extra control on suicide

Cell **123**, 49–63 (2005)

The p53 gene controls cell suicide and division, helping to stop cells becoming cancerous. After DNA damage, levels of the p53 protein increase. This happens because the protein is destroyed less quickly, but also, says Michael Kastan, because p53 translation increases. Kastan and his colleagues at the St Jude Children's Research Hospital in Memphis, Tennessee, found that ribosomal protein L26 binds to p53 messenger RNA and enhances its translation into protein. They also found a second protein, nucleolin, which inhibits p53 translation.

JOURNAL CLUB

Bruno Sicardy
Paris Observatory, and Pierre and Marie Curie University, Paris

An astronomer tells of the missing link in Saturn's rings.

The number of satellites known to be roaming between Saturn's rings and its moon Enceladus has soared over the past few decades as telescopes and instruments have improved. With the arrival of spacecraft, such as the current orbiter Cassini, satellites are even being discovered very near or inside the rings themselves.

Recently, Carolyn Porco of the Space Science Institute, Boulder, and her colleagues in the Cassini imaging team revealed six new moons in the ring region — typically no more than 5 km across (C. C. Porco *et al. Science* **307**, 1226–1236; 2005). Since this paper was published, a seventh moon has been detected in the narrow Keeler gap, which lies inside the A ring.

These new moons may hint at a missing link between the collisional, fluid-like rings and fully formed satellites. Some of them have a surprisingly low density, and their discovery has blurred the distinction between what is a satellite and what is merely a clumpy aggregate of dust.

Porco *et al.* estimated the density of some other moonlets by looking at the ripples they excite in the rings. For instance, Atlas, which orbits just 900 km outside the main rings, has a density of 0.5 g cm^{-3}. This is in the same ballpark as the values my colleagues and I obtained using Earth-based observations for the familiar moons, Prometheus and Pandora (S. Renner *et al. Icarus* **174**, 230–240; 2005).

The region just outside the main rings of Saturn is acting as a natural laboratory where we can admire short-lived, loose and fluffy aggregates as they emerge from their native rings. I expect that a continuous collision and re-accretion process transforms rings into satellites and vice versa with turnover times of a few tens of millions of years.