slices of rat brain and characterized the various lipid ions ejected from each pixel area. The resolution is lower than for related ionization imaging methods, but the sample needs no pretreatment and the images are obtained under ambient conditions, making *in vivo* use a possibility.

NANOTECHNOLOGY

Doubled up

Nano Lett. doi:10.1021/nl061898e (2006)

Fibres made with two semiconductors running side-by-side could be efficient devices for degrading organic pollutants, researchers report.

Zhaoyang Liu and Darren Delai Sun of Nanyang Technological University in Singapore and their colleagues used a technique known as electrospinning to make fibres about 100 nanometres wide that contained two thinner threads fused together. One of these was made from titanium dioxide (TiO_2), the other tin dioxide (SnO_2).

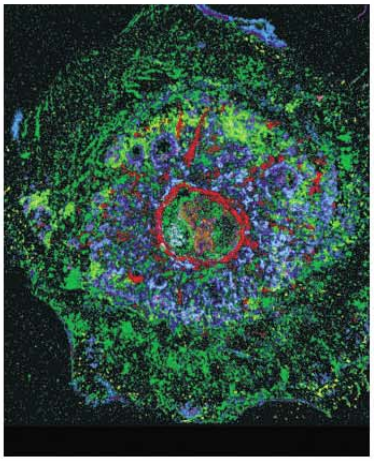
SnO_2 enhances the photocatalytic activity of TiO_2, which oxidizes organic materials using the energy from ultraviolet light. The twin fibre also has a greater surface area on which reactions can occur than other composite structures, such as bilayer films.

MICROBIOLOGY

Unclogging severe malaria

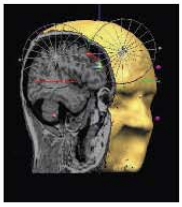
PLoS Pathogens **2**, e100 (2006)

Researchers in Sweden may have found a way to eliminate the nasty side effects of the malaria drug heparin. These side effects, which stem from the drug's anticoagulant activity, stopped its use.



Severe malaria occurs when red blood cells infected with the parasite *Plasmodium falciparum* stick to each other and to vascular walls. This causes a host of problems, including anaemia and respiratory difficulties. Heparin disrupts this binding, but can also cause severe bleeding.

A team led by Mats Wahlgren of the Karolinksa Institute in Stockholm has now shown that treating heparin with the compound periodate destroys its anticoagulant activity but retains its malaria-fighting properties. In tests on macaques, the modified drug was able to unstick infected cells and restore blood flow.



C. EISENEGGER

NEUROSCIENCE

Fair game

Science doi:10.1126/science.1129156 (2006)

Researchers have identified a brain region that has a critical role in our desire to punish unfair behaviour.

Daria Knoch and Ernst Fehr of the University of Zurich and their colleagues studied people playing the ultimatum game, in which one participant decides how to share a sum of money with a fellow player. If that player feels the offer is unfair, they can prevent the other participant from keeping any money by choosing to forfeit their own share.

When magnetic stimulation was used to inhibit activity in the brain's right dorsolateral prefrontal cortex, players become more likely to accept unfair offers.

PROTEOMICS

Mapping togetherness

Nature Biotechnol. doi:10.1038/nbt1250 (2006)

A method to map a hundred or more proteins in a single cell or tissue slice has been developed by Walter Schubert of the University of Magdeburg, Germany, and his colleagues.

They designed robotic workstations to carry out repeated rounds of antibody labelling and fluorescent imaging, identifying different proteins in sequence. A novel algorithm then analysed the images, pixel-by-pixel, to provide a map of protein networks in the cell or tissue (pictured left). The scientists applied the technique to compare protein distributions in diseased and healthy tissues.

It is hoped that such methods will help to unravel how the hundreds of thousands of proteins in a cell interact with each other, eventually in real time.

JOURNAL CLUB

Brent R. Stockwell
Columbia University, New York, USA

A chemical biologist is drawn to a new way of validating cancer drug targets.

I am interested in translating basic research into cancer therapies, so I am always looking for new approaches to selecting drug targets. Recently, I read of a technique that promises to help us cope with changing ideas about the types of target we should go after.

Typically, researchers have concentrated on finding proteins that trigger the genesis of tumours; the products of oncogenes. Some subsets of these 'oncoproteins' can be targeted by a class of drug known as small molecules.

My lab's drug-discovery strategy involves the use of synthetic lethal screens. We screen small molecules to identify those that are lethal to tumour cells with a specific oncogene.

I have noticed that, in tumour cells, effective small molecules often target proteins that are not products of oncogenes. These other proteins are not involved in tumorigenesis, but seem to be required for tumour maintenance.

In May, researchers reported a way of testing such 'tumour maintenance' genes in mice (K. Politi *et al. Genes Dev.* **20**, 1496–1510; 2006).

They used a switch known as an inducible promoter to turn a classic oncogene on and off. The oncogene, a mutant epidermal growth factor receptor (EGFR), triggers lung cancer by causing excessive cell proliferation. Turning off the gene in existing tumours in mice tested whether the mutant EGFR was also required for the tumour's maintenance. The team found that it was.

Although oncogenes are crucial to tumour formation, it is the tumour maintenance genes that we should target with therapeutic drugs. Here, we have a way to validate such targets that I encourage my colleagues in the translational cancer field to adopt.