are also thought to boost autoimmunity under certain circumstances by acting on other immune cells in the lymph nodes. Fu-Dong Shi at St Joseph's Hospital and Medical Center in Phoenix, Arizona, and his colleagues now report that NK cells have a stronger effect on autoimmunity in the central nervous system.

The researchers studied a mouse model of multiple sclerosis, a disease in which the immune system attacks myelin, the protective sheath surrounding nerve fibres in the brain and spinal cord. They found that NK cells limit inflammation and the immune response against myelin antigens. Increasing the number of NK cells in the central nervous system protected the animals from disease, whereas limiting the cells' numbers made symptoms worse. The researchers speculate that drugs used to treat multiple sclerosis may exert their positive effect by increasing NK cell numbers.

NEUROBIOLOGY

Autism detector

J. Neurosci. **30,** 10612–10623 (2010)

Brain scans may be sufficient to identify people with autism spectrum disorder (ASD), thanks to a new application of a type of data analysis.

Christine Ecker and her colleagues at the Institute of Psychiatry at King's College London scanned the brains of 20 adults diagnosed with ASD and 20 other volunteers using magnetic resonance imaging. The researchers' support vector machine (SVM) analysis — which is also used in face recognition — searched the data for subtle differences in cortex morphology between the two groups, using several parameters previously linked to ASD, such as cortical thickness and cortical folding. This identified several small, mostly non-overlapping, differences.

When participants were compared individually with data from the groups, SVM analysis identified ASD in as many as 90% of cases, which is comparable to the accuracy of behavioural diagnosis.

GEOSCIENCE

Ocean acid control

Geophys. Res. Lett. doi:10.1029/2010GL043181 (2010)

As atmospheric carbon dioxide levels rise, some of that gas dissolves in ocean waters, lowering the surface pH and potentially harming marine ecosystems. Quick and aggressive emissions reductions are key to minimizing this acidification, say Dan Bernie at the Met Office Hadley Centre in Exeter, UK, and his co-workers.

By coupling a climate model to ocean and terrestrial carbon models, the researchers simulated the effect of more than 100 emissions scenarios on ocean pH. The model outputs indicate that, without any mitigation strategy in place, global mean surface pH would drop from current levels of 7.9–8.3 to between 7.67 and 7.81 by 2100. But in an aggressive mitigation scenario, in which emissions peak in 2016 and then decrease by 5% each year, pH would end up at around 8.

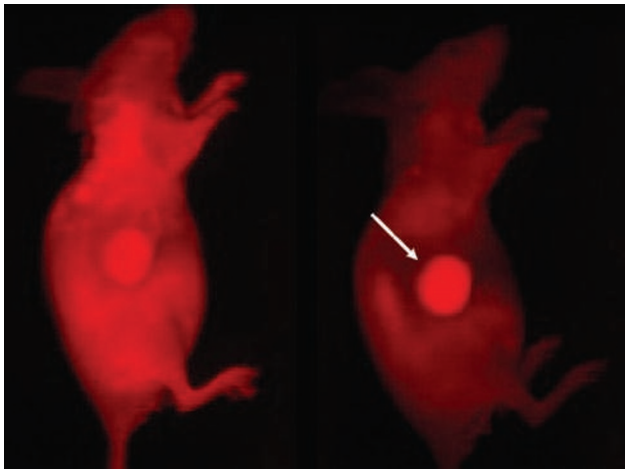
CANCER THERAPEUTICS

Nano tumour killer

Nano Lett. doi:10.1021/nl100996u (2010)

Potential weapons against cancer are not limited to small-molecule drugs, with nanomaterials such as carbon nanotubes among other candidates. The latest hot material, graphene — single-atom-thick sheets of carbon — seems to home in on tumours and, with the help of a laser, can heat up and kill them from within.

Zhuang Liu at Soochow University in Suzhou, China, and his colleagues coated nanometre-scale graphene sheets with polyethylene glycol to increase their solubility and stability in the body. They then injected the material into tumour-bearing mice (pictured left) and found high levels of graphene accumulation in their tumours after 24 hours (right).



AM. CHEM. SOC.

The team administered the graphene to another set of 10 mice with breast tumours and shone lasers at the growths. The tumours disappeared the following day and did not regrow during the 40-day experiment. Tumours in control mice that did not receive either the graphene or the laser treatment grew rapidly, killing the mice in about 16 days.

Although a small toxicity study did not reveal any obvious side effects, the authors say that more safety studies are needed.

JOURNAL CLUB

Gerry Melino
Medical Research Council, University of Leicester, UK

A cancer biologist weighs up p53, metabolism and cancer.

The classic tumour-suppressor gene, *p53*, plays a pivotal part in halting the cell cycle and inducing programmed cell death in response to DNA damage. However, recent data suggest that it also has a role in cellular metabolism. I have become intrigued by the possibility that the inactivation of p53, which is common in tumours, also contributes to a cellular shift from a metabolic pathway called oxidative phosphorylation to a less efficient one known as glycolysis. This shift, called the Warburg effect, is characteristic of tumour cells.

Two papers shed light on this possibility. Both show that GLS2, an enzyme involved in oxidative phosphorylation, is regulated by p53 under stressed and non-stressed conditions. Arnold Levine at the Institute for Advanced Study in Princeton, New Jersey, and his colleagues also show that GLS2 increases the respiration rate in the cell's energy-producing organelles, the mitochondria, resulting in increased generation of the cell's fuel source, ATP (W. Hu *et al. Proc. Natl Acad. Sci. USA* **107,** 7455–7460; 2010).

Meanwhile, Carol Prives at Columbia University in New York and her co-workers find that GLS2 expression is lost, or greatly decreased, in liver cancers, and that overexpression of GLS2 reduces the number of tumour cell colonies formed (S. Suzuki *et al. Proc. Natl Acad. Sci. USA* **107,** 7461–7466; 2010). The results reveal that GLS2 is an important component in mediating a novel function of p53: the regulation of energy metabolism.

This is an attractive and provocative hypothesis. There are some understandable discrepancies in the data, which suggests that additional mechanisms may be contributing to the metabolic changes. Nevertheless, these two papers provide a potential mechanism linking the metabolic and genetic characteristics of tumours.

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