Oceanography

Planktonic life on an ocean wave

Geophys. Res. Lett. **31,** L13301
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A wave doesn't sound like a terribly surprising thing to find in the ocean, but the one reported by Meric A. Srokosz and colleagues is. It is a wave of plankton growth, and it propagates for three months over a distance of about 2,500 km before dying out. The plankton wave shows up in remote sensing of chlorophyll in the Indian Ocean in early 1999; similar waves are apparent in data from 2000 and 2002.

The wave moves from west to east — against the mean ocean-surface flow, the direction of large-scale ocean waves (Rossby waves) and the direction of eddy propagation. So the pulse of plankton blooming isn't caused by advection. Srokosz *et al.* believe it may be a reaction–diffusion wave, like those seen in oscillating chemical reactions and other spontaneously pattern-forming systems. 'Reaction' here means phytoplankton growth, and 'diffusion' is the mixing caused by turbulent eddies. A rough estimate of the wave propagation speed in such a reaction–diffusion process matches that seen experimentally (about 10 km per day). **Philip Ball**

Chemistry

The grasping claws of DNA

Angew. Chem. Int. Edn **43,** 3550–3553 (2004)

A 'DNA machine' that can precisely control the concentration of the human blood-clotting factor, α-thrombin, does so by a novel grab-and-release mechanism, according to Wendy U. Dittmer and colleagues.

The machine is based on a known 15-base DNA sequence that adopts a chair-shaped conformation in the presence of potassium ions, which makes it bind strongly to α-thrombin. The authors modified this sequence with a 'switch' — an extra sequence of 12 bases that controls the binding ability of the entire strand. Under normal conditions, this switch does not affect the α-thrombin-binding ability of the strand. But a second DNA sequence can trigger the switch by binding to it; when added to the mixture, it destroys the machine's conformation and releases α-thrombin.

The molecular trigger can be removed from the mix by adding a DNA sequence that is fully complementary to it. This locks away the trigger, freeing up the switch and allowing the α-thrombin to be bound again. The DNA machine can bind and release α-thrombin through many cycles, and each step has a half-life of a few minutes. **Mark Peplow**

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Solid-state physics

Origin of electrical hum

Phys. Rev. Lett. **92,** 257202 (2004)

In the dead of night, our homes hum. The buzz emitted by domestic electrical appliances is the sound of a transformer flexing to the rhythm of the a.c. power supply, as magnetostriction creates tiny changes in volume. This effect has been poorly understood at a fundamental level, but now Th. Strässle *et al.* have identified its quantum origin.

An interaction called exchange coupling between the atoms of magnetic systems results from the spin of their electrons. This modifies the direct interaction via magnetic dipoles, so that in a periodically changing magnetic field the equilibrium distance between atoms also changes. In effect, the increase in elastic energy is compensated for by a reduction in magnetic energy. But it hasn't been clear whether this 'exchange striction' can really account for such volume changes.

Strässle *et al.* have measured the exchange energy by using inelastic neutron scattering to monitor the energy spectra of dimers of manganese ions at different pressures in the salt $CsMn_{0.28}Mg_{0.72}Br_3$, in which chains of Mg ions are punctuated by pairs of Mn ions. They measure the corresponding Mn–Mn distances using neutron diffraction. The observed exchange striction energy seems sufficient by itself to account for the changes in Mn–Mn excitation energy caused by squeezing. **Philip Ball**

Huntington's disease

Complex difficulties

Cell **118,** 127–138 (2004)

Huntington's disease, a fatal adult condition characterized by uncontrolled movements and dementia, is linked to a defect in the huntingtin protein that means the molecule contains repeated glutamine sequences. This abnormality results in dysfunction and death among neurons in the brain.

Using mouse cells, Laurent R. Gauthier and colleagues have tried to figure out the molecular causes of this process. They find that undamaged huntingtin enhances the transport of brain-derived neurotrophic factor (BDNF) along microtubules inside neurons. Without this factor, neurons in regions such as the striatum, which sustains the most damage in Huntington's disease, die off. The glutamine-repeat defect renders huntingtin unable to perform this function, probably because it cannot form a proper complex with the motor proteins that power microtubule transport.

What's more, the administration of wild-type huntingtin restores microtubule transport of BDNF, the authors report. This hints at a possible strategy to rescue brain cells if the disease can be diagnosed before too much damage occurs. **Michael Hopkin**

Developmental biology

From lung to intestine

J. Biol. **3,** 11 (2004)

In the metaplasias that sometimes portend cancer, cells in one part of the body take on the characteristics of those from another. Tadashi Okubo and Brigid L. M. Hogan have unearthed one of signals that convert lung cells into intestinal ones.

Okubo and Hogan first showed that, from around halfway through mouse gestation, budding lung tissues switch on the Wnt signalling pathway, which is implicated in numerous steps of organ growth and development. They then genetically engineered mouse embryos so that the level of Wnt signalling was boosted above normal in the growing lungs.

The transgenic embryos did not survive to birth and their lungs were missing many of the normal cell types. A survey of genes expressed in the organ showed that many genes normally active in pulmonary cells were suppressed and replaced by those normally active in cell types lining the intestine.

The result may explain the origin of particular forms of metaplasia in humans, such as premalignant stomach cancer or Barrett's oesophagus, when intestinal cells sprout out of place. The authors suggest that inflammation or injury might trigger stem cells to divide and to aberrantly switch on the Wnt pathway. **Helen Pearson**