

## Plant science

## Self-help for the interactive root

*New Phytol.* **157**, 315–326 (2003)

Plant roots don't just remove substances from their surrounding environment — they put things back, too, in the form of mucilage. From their investigations, D. B. Read and colleagues conclude that mucilage modifies the physical and chemical properties of soil, making it easier for plants to take up water and nutrients.

Read *et al.* analysed the composition of mucilage from the roots of maize, wheat and lupin. They found the chemical content to be very different from that in the plant tissues, implying that the plants were not merely leaking but were exuding substances for a specific purpose. Mucilage contains phospholipids, which are surfactants and which, like a detergent, reduce the surface tension of water. The authors tested surfactant action on soil. They found that it should enable plants to take up water from smaller pockets in the soil than they could otherwise do, and that it promotes the dissolution of phosphorus from soil particles into the water, increasing the availability of this nutrient by 10%. **John Whitfield**

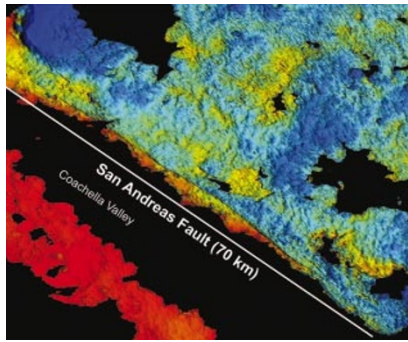
## Earth science

## Getting the measure of creep

*J. Geophys. Res.* **108**, doi:10.1029/2002JB001831 (2003)

When creep occurs at earthquake faults, it releases stress in the uppermost crust that would otherwise build up and, if suddenly released, would manifest itself as an earthquake. As creep movement is typically less than 10 millimetres per year, its measurement requires high precision. This has been achieved at only a few faults, using creep-meters or dense networks of global-positioning-system receivers. Suzanne Lyons and David Sandwell show, however, that creep can be accurately followed by drawing up interferograms (see picture) from data collected by satellites equipped with synthetic aperture radar. They believe that it is even possible to measure fault movement in regions covered in vegetation or in built-up areas.

Lyons and Sandwell used multiple satellite images of the southern San Andreas fault to automatically identify objects — such as buildings and outcrops of rock — that reflect radar consistently and so act as markers of creep. They further boosted sensitivity by layering the measurements to filter out transient phenomena such as turbulence in the atmosphere. The authors claim that the method can monitor creep over large areas



Displacement in southern California. Red and blue, respectively, show motion away from and towards the satellite. Regions without good point 'scatterers' are black.

and at higher spatial resolution than can the global-positioning system. **Tom Clarke**

## Genomics

## A bumper harvest of potato genes

*Plant Physiol.* **131**, 419–429 (2003)

The common potato (*Solanum tuberosum*) is the world's fourth largest food crop, with some 300 million tonnes grown annually. A member of the family Solanaceae, which also includes plants such as deadly nightshade and tomato, the potato has many similarities to its relatives. But it also has some outlandish characteristics, notably its tubers — the starch-filled root structures that are so versatile in cooking. C. Robin Buell and colleagues have now used genomics to investigate tuber development.

The group sequenced some 83,000 complementary DNAs from potatoes, representing nearly 20,000 genes; cDNAs provide an indication of which genes are active in a given tissue at a given time. The cDNAs came from four stages of tuber development: stolon, microtuber, dormant tuber and sprouting eyes. The authors thereby identified genes with a possible role in tuber development.

Potatoes are also prey to a notorious disease called potato late blight. By comparing cDNAs from uninfected and blight-infected leaves, and from plants either resistant or susceptible to blight, the group could pinpoint genes involved in resistance as well. **Christopher Surridge**

## Particle physics

## Photon charge reaches new limits

*Phys. Rev. D* **67**, 017701 (2003)

Photons have no electrical charge, according to conventional wisdom. But are we sure? A new measurement places an upper limit on the charge a photon might carry of

$8.5 \times 10^{-17}$  times the charge on an electron ( $e$ ). That is only a slight improvement on the previous limit of  $10^{-16}e$  from a lab measurement, but Y. K. Semertzidis and colleagues claim that their experiment could, with minor adjustments, achieve a sensitivity of  $10^{-21}e$ .

Astronomical observations of radiofrequency photons from a millisecond pulsar have previously placed a far more stringent constraint on the photon's charge:  $5 \times 10^{-30}e$ . But this figure relies on uncertain assumptions about the nature of intergalactic magnetic fields.

The new experiment is simple in principle. A laser beam is passed through a magnet and, after travelling 21 metres, is focused onto a photodiode. The magnet's field is modulated, and charged photons should exhibit this same modulation frequency, in phase with the magnet, as they arrive at the photodiode. The researchers see no sign of this modulation in the (noisy) photodiode signal. Using a longer flight path and faster modulation could increase the sensitivity by up to five orders of magnitude, the authors say. **Philip Ball**

## Cell biology

## Double trouble with mutant p53

*Mol. Cell* **11**, doi:10.1016/S1097276503000509 (2003)

In the event of a cellular emergency such as stress or DNA damage, the p53 protein can trigger apoptosis — the tidy demolition of the cell. This helps to prevent the uncontrolled cell proliferation that is a hallmark of cancer. Being a transcription factor, p53 is known to activate genes that are involved in apoptosis. But it has long been suspected of also having a more direct role in the cell's demise. Motohiro Mihara *et al.* now place p53 at the heart of the action — the cell's energy-producing organelles, the mitochondria.

Gene activation by p53 results in the perforation of mitochondrial membranes, the consequent leakage of apoptotic proteins, activation of specific enzymes and, ultimately, the shredding of proteins and DNA. Exactly how the mitochondrial membranes are perforated is not known, but the extended family of Bcl-2 proteins is intimately involved.

Mihara *et al.* now find that p53 binds two minders of mitochondria, Bcl-xL and Bcl-2. This prevents them from protecting mitochondria and results in the release of apoptotic proteins. Interestingly, tumour-derived p53 mutants that cannot bind DNA and stimulate transcription seem also to be unable to bind Bcl-xL. So, by blocking two routes to cell death, one mutation may mean double trouble. **Marie-Thérèse Heemels**