RESEARCH HIGHLIGHTS

CANCER GENOMICS

Prognostic sign

Nature Med. doi:10.1038/nm.2174 (2010)

A tumour's genetic make-up holds important clues to its stage of development, and researchers are now closer to a tool that can 'read' this information.

Soft-tissue cancers called sarcomas are normally staged, or graded, largely according to the tumour cells' appearance under the microscope. This method is more than 20 years old and is not very reproducible from one pathologist to the next. Frédéric Chibon at the Bergonié Institute in Bordeaux, France, and his co-workers have teased out a signature pattern of gene expression, involving 67 genes, that better predicts the five-year metastasis-free survival rate for people with sarcomas.

The researchers analysed gene-expression patterns for 183 sarcoma samples and tested their prognostic signature in a separate set of 127 sarcomas. Almost all of the 67 genes are involved in cell division or maintaining chromosome integrity. Further validation is needed before this can be used in the clinic, the authors say. **C.L.**

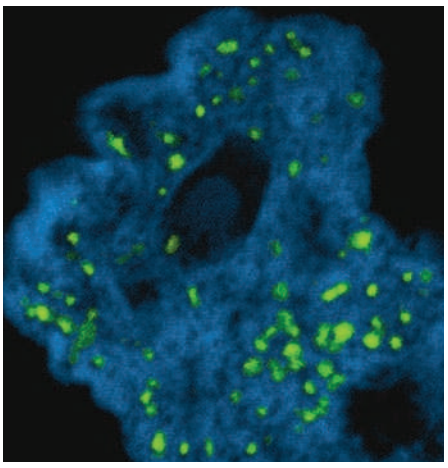
MICROBIOLOGY

Independent living

Mol. Microbiol. doi:10.1111/j.1365-2958.2010.07241.x (2010)

A group of bacteria that can multiply only in a host cell is less needy than scientists thought.

Members of the phylum Chlamydiae, which include human pathogens and symbionts of amoebae, were thought to be metabolically dependent on the cells they infect. Michael Wagner at the University of Vienna and his colleagues used Raman microspectroscopy to distinguish between the active and resting stages of the bacterium *Protochlamydia amoebophila* (pictured in green) on the basis of their chemical composition.



B. SIXT/UNIV. VIENNA

They expected resting bacteria to be metabolically inactive, but found that the cells took up the amino acid phenylalanine to produce proteins, even when outside their host cells. This uptake continued for up to three weeks, and the bacteria remained infective throughout. And the species was not alone: the human pathogen *Chlamydia trachomatis* also synthesized proteins independently of its hosts. **H.L.**

ASTRONOMY

No planetary X-ray pull

Astron. Astrophys. **515,** A98 (2010)

Planets seem to have no effect on the X-ray output of their parent stars, say Katja Poppenhaeger and her colleagues at the University of Hamburg in Germany.

Theorists have proposed that a planet's gravity could excite a star by causing it to bulge slightly or, similarly, that a planet's magnetic field could connect with that of its star and cause X-ray 'hotspots'. A previous study presented evidence for this effect.

Poppenhaeger *et al.* studied X-rays streaming from 72 nearby stars that host planets. In contrast to the earlier work, they find no obvious effect, and say that the phenomenon might appear in a few instances, for the most massive, close-in planets. **E.H.**

ECOLOGY

Don't damage dingoes

Ecol. Lett. doi:10.1111/j.1461-0248.2010.01492.x (2010)

Invasive species are often held responsible for driving native ones to extinction. Arian Wallach at the University of Adelaide in Australia and her colleagues propose that although invasives may drive biodiversity loss, a lack of ecosystem resilience is the ultimate cause of extinctions.

The authors assessed seven sites in Australia with differing levels of control of the top predator, the dingo (*Canis lupus dingo*; pictured). They found that harsh control actually promoted the spread of invasive species by disrupting dingo social structures and hence the resilience of the entire ecosystem. Relaxing control re-established biodiversity.

The team suggests that conservation managers should promote the inherent resilience of ecosystems rather than remove established invaders. **D.P.C.**

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IMMUNOLOGY

Gene plus virus

Cell **141,** 1135–1145 (2010)

Genes alone are rarely sufficient to cause disease. Researchers now report one possible explanation for this in Crohn's disease, a common inflammatory bowel disorder.

Thaddeus Stappenbeck and Herbert Virgin at Washington University in St Louis, Missouri, and their co-workers show that mice display some of the cellular abnormalities seen in Crohn's disease when they have a mutated Crohn's gene called *Atg16l1* and are also infected with a specific gut virus.

The results suggest that these abnormalities occur through mechanisms similar to those of Crohn's disease. The authors say that mutations in multiple genes combined with additional environmental factors may recreate the full range of Crohn's symptoms in mice. **C.L.**

GENETICS

Gene plus gene

Proc. Natl Acad. Sci. USA **107,** 10602–10607 (2010)

Hundreds of genetic variants are associated with complex diseases, but in most cases little is known about how the variants actually contribute to the disease.