RESEARCH HIGHLIGHTS

Expanding horizons

Astron. Astrophys. doi:10.1051/0004-6361:20054185 (2005)

The first results from the Supernova Legacy Survey support the idea that the expansion of the Universe is driven by a 'cosmological constant'. This massive project uses the brightness of supernovae to measure the expansion.

In the 1990s, observations of supernovae revealed that the Universe's expansion was accelerating, pushed by an unknown effect — dubbed 'dark energy' — that counteracts gravity.

Some theoretical models suggest that the density of dark energy can change over time. But the results of this survey, from 71 supernovae, show that this density has been constant to within 10%. By the time the survey ends in 2008, the sample should have swelled to 700 supernovae.

ANGLO-AUSTRALIAN OBSERVATORY/DMI

IMMUNOLOGY

Taken alive

Immunity **23,** 503-514 (2005)

Immune-system sentinels known as dendritic cells have a reputation for mincing any foreign proteins they meet. They show the remains to T cells, which help to coordinate the immune response. But now US researchers have found that dendritic cells can also capture their prisoners intact and haul them directly before antibody-producing B cells.

Raphael Clynes and his team at Columbia University in New York studied dendritic cells in mice. They found that, with the help of a receptor known as FcγRIIB, cells could swallow a protein and then regurgitate it with its three-dimensional structure intact. Maintaining the protein's structure is key to antibodies recognizing it. What's more, the dendritic cells actively sought out B cells, meaning that B cells do not rely on randomly meeting with microbes, as was once thought.

GENE THERAPY

A great escape

Nature Mater. doi:10.1038/nmat1524 (2005)

Researchers from the University of Tokyo report progress in tackling the biggest challenge in gene therapy — getting DNA into cells safely and effectively. They have developed a light-induced gene-delivery system that minimizes the toxic effects of the treatment by building the DNA and the light-sensitive compound into a single structure.

Large molecules such as DNA tend to be taken into cells by endocytosis, whereby the outer membrane of the cell pinches off, trapping the complex in a compartment known as an endosome. The photosensitive molecule helps the DNA to escape the endosome by disturbing the membrane when triggered by light. Wrapping the molecule around the DNA minimizes unwanted damage to other organelles and membranes.

The researchers in Japan, led by Kazunori Kataoka, tested their complex in cultured cells and *in vivo* in rats.

QUANTUM OPTICS

As tangled as can be

Phys. Rev. Lett. (in the press); preprint at www.archive.org/quant-ph/0507128

A pair of photons have been entangled as never before by Paul Kwiat of the University of Illinois at Urbana-Champaign and his team.

When the quantum states of two particles are entangled, you cannot make measurements on one of the pair without determining the state of the other. This is key to quantum information processing, for example in schemes for secure cryptography. The 'hyper-entangled' photons demonstrated by Kwiat's group may offer advantages in some quantum information-coding schemes.

The researchers use two crystals with nonlinear optical properties to generate photon pairs that are entangled in every possible way: in the quantum states describing their polarization, orbital angular momentum and emission times. In all, this produces entanglement in the 36 different quantum states the photons can adopt.

DEVELOPMENT

Talking about regeneration

Science **310,** 1327-1330 (2005)

Even when sliced up finely, planarian flatworms manage to regenerate themselves from every sliver. Yet they lose this remarkable ability if just one of their genes, *smedwi-2*, is blocked. Researchers have found that blocking the gene prevents the worm's stem cells from maturing into adult cells capable of replacing ageing or missing cells.

The *smedwi-2* gene has counterparts in many species, and the team led by Peter Reddien, at the Whitehead Institute in Cambridge, Massachusetts, concludes that *smedwi-2* may be part of a universal regulatory mechanism for regeneration based on stem cells. In the planarian flatworm pictured below, black regions indicate *smedwi-2* expression.

P. W. REDDIEN & A. SÁNCHEZ ALVARADO

PHYSIOLOGY

Insects kick up a fuss

J. Exp. Biol. **208,** 4451–4466 (2005)

A new insecticide, which only affects plant-sucking bugs, was thought to target their central nervous system, as do many insecticides. But it seems that pymetrozine goes after the insects' chordotonal organs, according to Harald Wolf and his team from the University of Ulm in Germany and the Syngenta Crop Protection AG in Switzerland. These organs control the insect's joints, which explains why, in locusts (pictured right), dosed bugs kick up their heels and take on an unusual posture. How this relates to the functional effect of the insecticide — a cessation in feeding behaviour — remains unclear, as does the compound's molecular target.

IMAGE UNAVAILABLE FOR COPYRIGHT REASONS

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EVOLUTION

Carping on

Proc. R. Soc. Lond. B doi:10.1098/rspb.2005.3343 (2005)

Changing body shape to avoid predators can be costly in terms of the ability to compete for food, research on crucian carp (*Carassius carassius*) suggests.

Carp raised in the presence of chemical cues from pike developed a body shape with a greater depth than those growing up without the fear of predation. Similar changes were observed when differing food types were offered, show Jens Andersson and his colleagues at Umeå University in Sweden. Those that were fed on bottom-dwelling chironomids also adopted a deep body shape, whereas those fed on tiny swimming zooplankton had a shallower body.

When offered zooplankton later in the experiment, those carp that had become deeper as a result of either being fed on chironomids or being raised with pike cues were less adept at feeding than their shallow-bodied counterparts.

STEM CELLS

Grow your own liver

Nature Biotech. doi:10.1038/nbt1163 and 10.1038/nbt1167 (2005)

Making embryonic stem cells turn into liver or pancreas cells should get easier with new methods for producing endoderm — the embryonic germ layer from which liver and pancreas are derived.

Emmanuel Baetge's group at CyThera in San Diego, California, showed that a culture rich in activin A encouraged human embryonic stem cells to differentiate into endoderm, achieving an 80% success rate.

In Japan, a team led by Shin-Ichi Nishikawa of the RIKEN Center for Developmental Biology in Kobe also used activin to induce mouse embryonic stem cells to differentiate into endoderm 25% of the time. Furthermore, they created a monoclonal antibody to monitor the differentiation.

CELL BIOLOGY

Wrinkles in theory of ageing

Cell **123,** 655–667 (2005)

The protein Sir2 has been shown to prolong life in some species, such as worms and flies, but its action in yeast hints at an undiscovered complexity in the mechanism.

If yeast ageing is measured in terms of how many daughters a cell can produce, Sir2 boosts longevity by 40%. But Valter Longo at the University of Southern California in Los Angeles and his colleagues show that, if the chronological age of the cells is measured, Sir2 limits a yeast's lifespan. The team found that overexpressing the *SIR2* gene in long-lived yeast mutants hastened their demise. They also showed that yeast lacking *SIR2* had reduced rates of DNA mutation and more active stress-resistance genes.

MOLECULAR BIOLOGY

Evasion tactics

J. Exp. Med. **202,** 1319–1325 (2005)

A compound that protects parasitic eggs from their host's immune system could one day form an anti-inflammatory drug for humans, researchers suggest.

Padraic Fallon of Trinity College in Dublin, Ireland, and his colleagues investigated how the parasitic flatworm *Schistosoma mansoni* evades immune detection. The team knew that certain viruses make 'chemokine-binding' proteins, which bind and neutralize protective immune compounds. The researchers succeeded in isolating this type of protein from *S. mansoni* eggs. They also showed in mice that the protein could suppress skin inflammation and prevent chemokine-induced lung inflammation.

JOURNAL CLUB

Guy Salvesen
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A cell biologist explains why his colleagues shouldn't look down on cleavages.

If I had a professional mantra it would be, "Don't inhibit the protease that is cleaving your favourite protein; find out what the function of the cleavage is."

Some cell biologists go to great lengths to avoid proteolysis — or breaking up — of the transcription factors, signalling kinases or cell-surface glycoproteins on which they work. But they could get to love the enzymes that do the snipping, when they realize that the cleavage often defines important aspects of cell signalling.

At my lab we spend a lot of time discovering how cellular processes are governed by proteolysis, and trying to find out how misregulation of proteolytic signalling leads to, for example, degeneration and cancer.

There is now an emerging theme in the field, supported by recent key papers, which suggests that proteolytic activity in the wrong place can change the nature of cells. I have wondered whether a misplaced protease, causing abnormal activation or inactivation of a signalling protein, could promote cell growth and so cause cancer.

Several proteases have been implicated in cancer, but always in a rather roundabout way — through observations of how they influence the progression of tumours implanted in mice, for example.

So I was intrigued to read about a cell-surface protease that can directly cause skin cancer in mice when its expression is abnormally forced in the epithelium (K. List *et al. Genes Dev.* **19,** 1934–1950; 2005). Because overexpression of the protease in question, matriptase, is correlated with a variety of epithelium-derived tumours in humans, there is finally a chance to understand how mislocation of a protease causes cancer. There is even the prospect of a therapy.