

those with normal autophagy. Moreover, autophagy dysfunction seems to be associated with activation of inflammasomes — protein complexes that trigger inflammation — in the macrophages.

A team led by Ira Tabas at Columbia University in New York has found that mice that had defective macrophage autophagy and ate a fatty diet showed more signs of advanced atherosclerosis, including plaque necrosis, which has been linked to heart attacks and stroke in humans. *Cell Metab.* <http://dx.doi.org/10.1016/j.cmet.2012.02.011>; <http://dx.doi.org/10.1016/j.cmet.2012.01.022> (2012)

MATERIALS

Solvent-free 'ink' glows white

Low-cost, light-emitting inks that can be painted onto surfaces could be a boon for large electronic displays. Researchers have come up with a method of producing glowing ink that avoids the use of solvents, which typically must be evaporated away after the ink is 'printed'. This evaporation can change the ink's colour.

Takashi Nakanishi at the National Institute for Materials Science in Tsukuba, Japan, and his colleagues synthesized small organic molecules that form luminescent liquids at room temperature. These were mixed with other dyes to form a stable paste that glows in shades of white when illuminated with ultraviolet radiation, as illustrated by a light-emitting diode (pictured, left) painted with the paste (right).



PRISMA BILDAGENTUR/ALAMY

WILEY-VCH VERLAG

The new paste produces an impractical sticky film, but the researchers say they are working to solve the problem. *Angew. Chem. Int. Ed.* 51, 3391–3395 (2012)

CANCER

Tumours yield to pressure relief

Lethal pancreatic tumours can be rendered sensitive to chemotherapy by degrading a sugar-based matrix in the tumour that boosts fluid pressure and prevents the inward flow of blood.

Sunil Hingorani at the Fred Hutchinson Cancer Research Center in Seattle, Washington, and his team show that pancreatic tumours in mice have much higher fluid pressures than healthy mouse pancreases owing to high levels of a sugar-based polymer produced by the tumours. An enzyme that chews away the matrix returns fluid pressure to normal, opening up the blood vessels that feed the tumour and allowing chemotherapeutic drugs to penetrate.

Mice treated with a combination of the enzyme and a cancer drug developed fewer tumours and metastases and lived longer — 91.5 days compared with 55.5 — than animals treated with the drug only.

Cancer Cell 21, 418–429 (2012)

ECOLOGY

Noise nixes seed spread

Noise pollution not only alters the behaviour of individual animals, it can also have a wider impact on ecosystems.

Clinton Francis at the National Evolutionary Synthesis Center in Durham, North Carolina, and his colleagues studied how noise levels affect the hummingbird *Archilochus alexandri* and the jay *Aphelocoma californica* (pictured) in an area of New Mexico that has a large

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BIOCHEMISTRY

Proteins' ticket into the cell

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Protein-based drugs are notoriously difficult to deliver into their target cells. Chemists at the University of Wisconsin-Madison have boosted the uptake of a protein by mammalian cells by attaching boronic acid groups to the protein.

Ronald Raines and his team homed in on a modified boronic acid, benzoxaborole, because it has a strong affinity for a sugar molecule abundant on cell surfaces.

The team attached the boronate groups to a protein, RNase A. This enzyme kills the cells that it enters, providing a visible measure of successful entry. Hamster cells internalized the boronated protein four to five times faster than the non-boronated version.

J. Am. Chem. Soc. 134, 3631–3634 (2012)

number of gas wells. Increased noise from wells equipped with noisy compressors boosted the pollination of artificial flowers by the hummingbirds, but lowered the number of jays, which disperse pinyon (*Pinus edulis*) seeds. At the noisier sites, the numbers of pinyon seedlings were just



one-quarter of those recorded by the authors at quiet sites. The authors link this shortage to the relative scarcity of jays. *Proc. R. Soc. B* <http://dx.doi.org/10.1098/rspb.2012.0230> (2012)

that makes chilli peppers hot — and its receptor.

Richard Palmiter at the University of Washington in Seattle and his colleagues genetically modified mice so that they expressed a cell-surface receptor for capsaicin only in brain neurons that release the neurotransmitter dopamine, and not in the neurons of the peripheral nervous system that are responsible for the painful response to capsaicin.

When the researchers fed or injected the mice with capsaicin, they found that the compound activated brain dopamine neurons only and stimulated behaviour consistent with increased dopamine release. Similar results were seen in mice with the capsaicin receptor present only on cells that release another neurotransmitter, serotonin.

The researchers could reverse and repeat the effects of capsaicin, as the compound activated neurons for no more than 10 minutes after each administration and did not injure the mice.

Nat. Commun. 3, 746 (2012)

NEUROSCIENCE

Chilli compound triggers neurons

Neuroscientists have various ways of activating specific neurons in animal brains to determine the cells' roles in neural circuits. A twist on these techniques that its inventors say is less labour-intensive and invasive involves capsaicin — the compound

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