showed poorer recovery of blood flow than those receiving a sham treatment — an effect that was also reversed by DHT.

ASTRONOMY

Hot spectra

Astrophys. J. **710,** L35–L38 (2010)

Astronomers have for the first time directly captured the spectrum of light emitted by a planet orbiting a Sun-like star outside the Solar System. Space telescopes have previously measured spectra from planets beyond the Solar System — a way to learn about their atmospheres — although this could only be done in the rare cases in which a planet was in front of or behind its star in relation to Earth.

Markus Janson of the University of Toronto in Canada and his colleagues needed almost six hours of time on the Very Large Telescope in Chile to obtain the direct spectrum of the young, massive and hot planet called HR 8799c. It doesn't match modelled spectra for a typical hot planet, suggesting that HR 8799c might be surrounded by dust or that its atmosphere is still mixing turbulently.

NEUROBIOLOGY

Prions at work

Nature Neurosci. doi:10.1038/nn.2483 (2010)

Misfolded prion proteins can cause neurodegenerative disease, but what purpose normal prions serve has been hard to pin down.

Now Adriano Aguzzi at the University Hospital of Zurich in Switzerland and his team show that the proteins are needed to maintain the myelin sheath that surrounds nerves and ensures that they function properly.

In the group's experiment, mice from four strains that were deficient in the prion protein all showed signs of neuropathy associated with demyelination. The damage was prevented when the protein was reintroduced to nerve cells but, surprisingly, not when it was reintroduced to Schwann cells, which produce myelin. Only variants of prion proteins that could be cleaved by enzymes could rescue the neuropathies, suggesting that enzyme processing of the prion is required for myelin maintenance.

The authors say that prion fragments may interact with receptors on Schwann cells to maintain the myelin sheath.

For a longer story on this research see go.nature.com/zK5sEq

MODIS RAPID RESPONSE TEAM, NASA GODDARD SPACE FLIGHT CENTER

ATMOSPHERIC SCIENCE

Stronger storms

Science **327,** 454–458 (2010)

Atlantic hurricanes might become fewer but fiercer in the Bahamas and the southeastern United States as a result of global warming.

Morris Bender at the National Oceanic and Atmospheric Administration in Princeton, New Jersey, and his team used a high-resolution operational hurricane model together with 18 global climate models to project the influence of rising sea surface temperatures on future hurricane activity in the western Atlantic.

The analysis suggests that the frequency of major hurricanes (categories 4 and 5) will roughly double by the end of the century, whereas the overall number of tropical storms and hurricanes will decrease by about one-third.

For a longer story on this research see go.nature.com/RN5AIx

CANCER BIOLOGY

Weighted cancer risk

Cell **140,** 197–208 (2010)

Obesity is a contributor to cancer. Work in mice now reveals the mechanism by which obesity enhances inflammation and tumour growth in the liver.

Michael Karin and his co-workers at the University of California, San Diego, found that both mice eating a high-fat diet and those genetically engineered to be obese had higher incidences of liver cancer and had larger and more numerous tumours than normal mice when given a known carcinogen.

Obese animals had higher levels of activated STAT3, a known cancer-promoting protein. Obese mice also had elevated levels of IL-6, an immune-modulating and tumour-promoting protein, and TNF, a proinflammatory protein. The team found that both IL-6 and TNF, which activate STAT3, are required for obese mice to develop inflamed livers, which are at greater risk of becoming cancerous.

JOURNAL CLUB

Jay Shendure
University of Washington, Seattle

A geneticist discusses a way to assess the effects of disease-causing gene mutations.

Although thousands of rare inherited disorders are clearly monogenic — caused by single-gene mutations — the overall picture is usually more complex. Genetic and environmental modifiers, as well as differences in the gene variants themselves, can affect how disease genes are expressed and how a disease manifests itself (the phenotype).

Marc Vidal of the Dana-Farber Cancer Institute in Boston, Massachusetts, and his team reveal that disease-causing mutations may fit into two groups on the basis of the type of perturbation they cause. 'Edgetic' mutations affect specific interactions in a network of genes, whereas 'nodal' ones remove proteins from the network altogether (Q. Zhong *et al. Mol. Sys. Biol.* **5,** 321; 2009). The researchers did computational analyses, using the tendency of disease-associated mutations to be in-frame — producing full-length mutated proteins — or truncating, producing truncated proteins, as a proxy for edgetic or nodal perturbations, respectively. They found that, for many genes underlying multiple diseases, different phenotypes were associated with different ratios of in-frame versus truncating mutations.

The authors then did experiments evaluating whether disease-associated mutations tend to disrupt known protein–protein interactions in a way that is consistent with edgetic versus nodal perturbation. They suggest that at least some of the phenotypic variability in monogenic diseases might correlate with specific patterns of network perturbation.

The experiments are limited, but the approach of cloning mutations and serially evaluating their impact is appealing. Various genome-sequencing projects will soon catalogue hundreds of thousands of coding variants of uncertain significance. Generalized, scalable methods to evaluate the functional relevance of these variants and to place them into a broader biological context will be crucial.

Discuss this paper at **http://blogs.nature.com/nature/journalclub**