atmosphere–ocean global circulation model that included two-dimensional representations of vegetation surfaces and soils to track the movement of DDT. Their analysis revealed that the western North Atlantic ocean has been re-emitting DDT for more than three decades, longer than most other regions. The distribution of the pesticide has been creeping northwards, despite the DDT bans in the northern high and mid-latitudes. The authors caution that DDT-monitoring data have not been gathered for long enough to test the model.

For a longer story on this research, see go.nature.com/ZVw7VN

IMMUNOLOGY

Double punch for HIV

J. Exp. Med. doi:10.1084/jem.20091933 (2009)

The antiviral enzyme APOBEC3G inhibits HIV infection by causing damaging genetic mutations in replicating viruses. Arnaud Moris of the Pasteur Institute in Paris and his colleagues now demonstrate that this enzyme also combats HIV by triggering the activation of the immune system's cytotoxic T cells, which kill helper T cells infected with HIV.

Moris and his co-workers showed *in vitro* that HIV particles harbouring APOBEC3G are more effective activators of HIV-specific cytotoxic T cells than viruses lacking the enzyme. Mutating the enzyme's active site eliminated this effect.

NEUROSCIENCE

Dark migraine relief

Nature Neurosci. doi:10.1038/nn.2475 (2010)

For many migraine sufferers, light makes the pain worse. Rami Burstein of Harvard Medical School in Boston, Massachusetts, and his colleagues began their quest to figure out the neural mechanism behind this by studying 20 blind migraine sufferers. Those who could detect light reported heightened pain when exposed to it.

The researchers then examined the posterior thalamus — the brain region containing neurons that fire during migraines — of anaesthetized rats. They found that projections from a specific group of retinal cells converge on other cells that process both migraine pain and light signals. Most of the retinal cells involved can respond to light but cannot form images and are still functional in some blind people.

MOLECULAR BIOLOGY

Flowering time unravelled

Cell **140,** 136–147 (2010)

Plants are sensitive to temperature and respond to changes by, for example, adjusting flowering times. Vinod Kumar and Philip Wigge of the John Innes Centre in Norwich, UK, have discovered the molecular mechanism behind this in the model plant *Arabidopsis thaliana*. Warmer temperatures trigger the loosening of certain nucleosomes — tightly wound DNA structures — allowing the up- or downregulation of certain genes.

The researchers first identified a mutant (pictured above right) in which flowering occurs more rapidly than usual — the same behaviour seen in plants exposed to warmer temperatures. They found that the mutant plant is unable to incorporate a protein called histone H2A.Z into its nucleosomes to keep them wound up. The duo found that as temperatures rise, H2A.Z is less able to bind to nucleosomes, causing them to unravel.

DRUG DISCOVERY

Virtual antibiotic screen

Proc. Natl Acad. Sci. USA doi:10.1073/pnas.0909181107 (2010)

The hunt for new antibiotics can be aided by computational tools that identify indispensible components of a pathogen's physiology.

Olaf Wiest at the University of Notre Dame in Indiana, Zoltán Oltvai of the University of Pittsburg in Pennsylvania and their colleagues analysed the metabolic networks of *Escherichia coli* and *Staphylococcus aureus* and identified 13 enzymes that catalyse essential reactions in both these bacteria. The researchers then ran molecular simulations to find 41 compounds with potential to inhibit these enzymes. Finally, they confirmed that some of those compounds reduced enzyme activity and killed the bacteria *in vitro*.

The study, the authors say, shows how genomic and metabolic information can enhance drug discovery, including the development of tailored therapies for specific bacterial strains.

ELSEVIER

Correction

The two images illustrating the Research Highlight 'Dual-aspect particles' (*Nature* **462,** 828; 2009) were inadvertently swapped. The left-hand image shows the liquid phase of Janus particles, and the right-hand image shows the gas phase.

JOURNAL CLUB

Monica Gotta
University of Geneva Medical School, Switzerland

A cell biologist connects her research to bacterial brain invasion.

My main interest is in understanding how some cells organize their structure and components asymmetrically — a property called cell polarity. When I moved to my current job in a medical faculty I was asked to teach a course on infectious diseases. So I was very excited by the publication of a paper from Mathieu Coureuil at the University of Paris Descartes and his colleagues that brings together my passion and my teaching activity. The work shows that a bacterial pathogen can reach the brain by destroying cell polarity (Coureuil, M. *et al. Science* **325,** 83–87; 2009).

Few bacteria are able to cross the blood–brain barrier, and it is not known whether those that can do so by moving through or between cells. The bacterium *Neisseria meningitidis* can cross this barrier. It adheres to cells lining the brain's blood vessels using type IV pili — hairlike appendages that connect the bacterium to the interior of these endothelial cells.

Using human brain endothelial cells and *N. meningitidis* in culture, Coureuil *et al.* show that a complex of polarity proteins — Cdc42, PAR6, PKC and PAR3, which form tight junctions between endothelial cells — are recruited to the site of bacterial adhesion. This results in depletion of these proteins at the junctions and thus the formation of gaps between infected cells.

Although this study was performed in cultured cells owing to a lack of suitable animal models, it strongly suggests that *N. meningitidis* enters the brain by disrupting the junctions between cells — allowing the bacteria to squeeze in between them — and not by penetrating the cells themselves.

This elegant paper unveils a route that may also be used by other pathogens that cross the blood–brain barrier. It also underscores an important function of cell polarity: protecting our brain from infectious diseases.

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