

## CLIMATE CHANGE

## Warming boosts invasions

*PLoS ONE* 5, e8878 (2010)

Climate warming could exacerbate species invasions and their often-negative ecological impacts if non-native plants continue to respond better to changing conditions, a study warns.

Using a 150-year record of seasonal plant data started by US poet and naturalist Henry David Thoreau, Charles Davis at Harvard University in Cambridge, Massachusetts, and his colleagues analysed the long-term changes in the flowering times of native and non-native plants in Concord, Massachusetts, near where Thoreau lived.

The average yearly temperature in Concord has increased by 2.4°C since the 1850s. Invasive species have adapted more quickly to these changes in temperature, shifting their flowering time an average of 11 days earlier than native plants over the past 100 years. For example, the non-native mayweed chamomile now flowers 23 days earlier, whereas some natives are still flowering at the same time of year as in 1900, potentially putting them out of sync with their pollinators.

## STEM CELLS

## Uneven divide

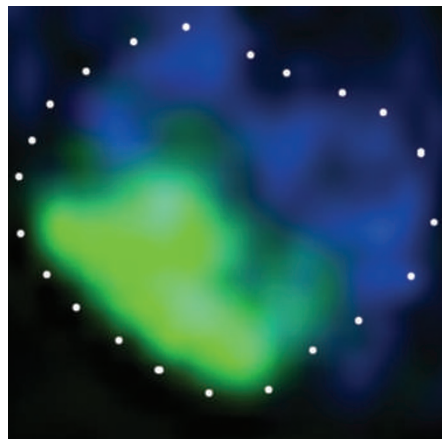
*Cell Stem Cell* 6, 175–181 (2010)

Loss of the directional asymmetry of cell division seen in certain stem-cell populations may give rise to cancer.

Inke Nätke of the University of Dundee, UK, and her colleagues imaged mitotic spindles — the structures that pull replicated DNA apart during cell division — in three dimensions in mouse and human intestinal cells. In the stem-cell compartments, the

spindles often oriented perpendicularly to the cell surface facing the intestinal cavity. This arrangement correlated with an asymmetrical distribution of DNA (pictured in green below) during cell division.

But in mice with a mutation linked to colorectal cancer, the spindles in the precancerous intestinal cells showed no bias towards a perpendicular orientation or asymmetrical division. The results suggest that loss of asymmetrical division in these cells contributes to the development of cancer.



## CARDIOVASCULAR BIOLOGY

## Fatty foam cells

*Cell Metab.* 11, 125–135 (2010)

A key element of heart disease is the accumulation of immune cells called macrophages that are filled with cholesterol, which gives the cells a foamy appearance. Researchers have identified a network of 46 proteins in macrophages whose regulation is linked to the conversion into ‘foam cells’, which contribute to atherosclerosis — the thickening of artery walls.

Jay Heinecke and his colleagues at the

University of Washington in Seattle used mass spectrometry to identify 777 proteins in macrophages. They then narrowed down the list to the 46 proteins that exhibited a notable increase or decrease in levels when macrophages became foamy. They mapped the proteins into a network by mining protein-interaction databases.

The authors found that regulation of this network was altered in cells treated with drugs that target heart disease, and that apolipoprotein E, a protein known to block atherosclerosis, is a key regulator of the network.

## GEOENGINEERING

## Ocean beating

*Geophys. Res. Lett.* doi:10.1029/2009GL041961 (2010)

In 2007, geoscientists proposed that artificial upwelling of nutrient-rich ocean water could help sequester a fraction of excess atmospheric carbon dioxide. The installation of large vertical pipes in suitable ocean regions would bring deeper water to the surface, enhancing photosynthesis.

Andreas Oschlies of the Leibniz Institute of Marine Sciences in Kiel, Germany, and his colleagues have now used a three-dimensional carbon-climate model to simulate the effect this might have on carbon fluxes. They found that under the most optimistic assumptions, the technique could sequester 900 million tonnes of carbon per year — about 10% of current annual emissions.

However, if artificial upwelling was later stopped, the model predicts that CO<sub>2</sub> concentrations and sea surface temperatures would rise to levels higher than those in a reference simulation in which artificial upwelling had never been undertaken.

## JOURNAL CLUB

**Paul Flicek**  
European Bioinformatics  
Institute, Cambridge, UK

**A computational geneticist  
looks at mechanisms of  
chromosomal evolution.**

Humans are unique among great apes in having 23 pairs of chromosomes, a result of the fusion of two ancestral chromosomes: chimpanzees and other great apes have 24. Although the differences between the human and chimp genomes greatly exceed this one

event, I find it hard to resist the idea that this single, major change kicked off an evolutionary process that eventually led to *Homo sapiens*.

The evolution of karyotype — the name given to a species’ collection of chromosomes — is normally a slow process. Although mammals exhibit an inter-chromosomal shuffling event on average once every 4 million years, not all primates have exhibited such restraint. Gibbons, humans’ closest relatives beyond the great apes, have a rate of chromosomal rearrangement that is 20 times higher than that of other primates.

So why does this reorganization happen so much more often for some species than others?

Lucia Carbone of the Children’s Hospital and Research Center Oakland in California and her team have proposed an intriguing answer. They analysed the human and gibbon genomes and found specific sites at which gibbons had lower levels of DNA methylation (the addition of methyl groups to DNA) than humans (L. Carbone *et al.* *PLoS Genet.* 5, e1000538; 2009). The researchers theorize that this lack of methylation led to the rapid evolution of the gibbon

karyotype by creating more open regions of the chromosome, which are more likely to recombine with other genetic elements.

This result could affect our understanding not only of genome evolution, but also of the pathogenesis of diseases such as cancer. The fundamental methylation mechanisms behind rapid chromosomal evolution may also be linked to the karyotype disruptions that are associated with some cancers.

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