C. compressirostris females a choice between males of the same species, and either a closely related but electrically different *C. rhynchophorus*, or a distantly related but electrically similar *C. tamandua*. The procedure was repeated with a simulation of the males' electrical discharges in place of the male fishes.

The electric signal seems to be all-important. In both tests, females preferred their own kind over *C. rhynchophorus*, but not over *C. tamandua*.

PHARMACOLOGY

Setting the pace

Cell Metab. **8,** 482–491 (2008)

A protein targeted by some diabetes drugs might also help to regulate daily cycles in blood pressure and heart rate.

Thiazolidinediones are widely prescribed for the treatment of type 2 diabetes, and work by activating a protein called PPAR-γ. Tianxin Yang of the University of Utah in Salt Lake City and his colleagues investigated the cardiovascular role of this protein using mice that lacked expression of the gene that encodes PPAR-γ in vascular smooth-muscle cells.

Rhythmic variations in blood pressure and heart rate, as well as in the expression of several body-clock genes in blood vessels, were diminished in these mice. Furthermore, normal mice treated with a thiazolidinedione called rosiglitazone showed increased expression of *Bmal1*, a clock gene.

QUANTUM PHYSICS

Signature shift

Science **322,** 1357–1360 (2008)

The discovery of the Lamb shift in 1947 was a key factor in the development of quantum electrodynamics (QED). This slight discrepancy between the predicted and observed energy levels of electrons in a hydrogen atom was eventually explained by QED as the effect of virtual photons flickering in and out of existence in a vacuum. Now that effect has been replicated in an electrical circuit by Andreas Wallraff at ETH Zurich in Switzerland and his colleagues.

Their work uses 'artificial atoms', quantum devices designed to have an energy spectrum resembling that of real atoms. Tuning a coupling between such artificial atoms and a microwave transmission line containing a strong vacuum allowed them to produce a Lamb shift of 1.4% for a typical emission line.

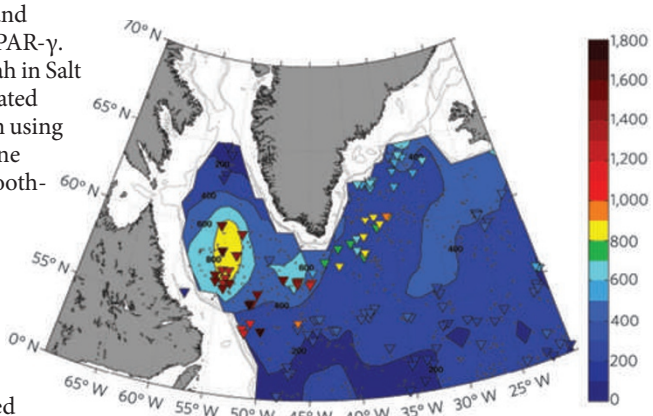
GENOMICS

The baby-milk bacterium

Proc. Natl Acad. Sci. USA **105,** 18964–18969 (2008)

The guts of breast-fed babies often contain bacteria of the subspecies *Bifidobacterium longum infantis*, which keep at bay harmful microorganisms that might otherwise take up residence. The genome of this subspecies has now been published.

Of its 2,423 recognized protein-coding genes, 702 are not found in related bacterial taxa sequenced so far, write David Mills of the University of California, Davis, and his colleagues. The bacterium feeds on specific sugars in human milk that infants do not use, and Mills and his team have identified several genes that make this possible. Another gene cluster makes enzymes that allow the microbes to consume milk-borne urea in the nitrogen-poor environment of the infant bowel.



GEOSCIENCES

Deep-sea mix

Nature Geosci. doi:10.1038/ngeo382 (2008)

After more than a decade of shallow or non-existent convection currents in the Labrador Sea — with only a brief return to deep-water convection at the turn of the century — the carbon-dioxide-sequestering subpolar gyre in the North Atlantic Ocean seems to have bounced back.

Kjetil Våge of the Woods Hole Oceanographic Institution in Massachusetts and his colleagues report data from the system of floats known as the Argo programme. These show a return last winter to mixing as deep as 1,800 metres in the Labrador Sea, 1,000 metres in the Irminger Sea and 1,600 metres south of Greenland. Triangles in the image (above) indicate these depths (in metres).

The change happened surprisingly quickly. The reasons for it include unusually low air temperatures, an increased flux of fresh water and pack-ice from the Arctic and changes in the North Atlantic storm track.

JOURNAL CLUB

John Greally
Albert Einstein College of Medicine, New York City

A biologist considers a link between jumping genes and immune-system enzymes.

Many viruses present a fierce threat to the body. They contain nucleic acids that, when free to roam in a cell's cytoplasm, elicit an immune response involving proteins called interferons. Pairings of the nucleic-acid residues cytosine and guanine are especially good at this, unless they carry a chemical modification in the form of a methyl group. This modification is the norm for 'jumping genes', or retrotransposons, which can move around the human genome and were probably once viral genes themselves.

A team led by Daniel Stetson at the University of Washington in Seattle has uncovered a useful twist to this tale. While searching for proteins that interact with cytoplasmic nucleic acids, the researchers came across Trex1. Mutated versions of Trex1 are known to cause chilblain lupus in humans, and in mice lead to autoimmune myocarditis, whereby the immune system attacks the heart. Stetson *et al.* say that mice lacking Trex1 have huge numbers of retrotransposons in their heart muscles.

Critically, the authors' molecular surveys reveal that Trex1 suppresses the rate at which jumping genes move around. This indicates that Trex1 protects the body from misidentifying its own parts as 'foreign' by degrading retrotransposons and thus preventing them from overloading the system (D. B. Stetson *et al. Cell* **134,** 587–598; 2008).

That jumping genes have the potential to overwhelm the system in this way was unexpected. Most experts had assumed that the addition of methyl groups took care of quenching them. But if retrotransposons are made at a rate that triggers inflammation, as Stetson and his colleagues' experiments propose, it could open up a whole new avenue for research. Everyone studying lupus and related diseases should be excited.

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