twentieth century, were from a different lineage altogether.

Because of the bacteria's rapidly evolving genome, the researchers say that cholera strains should be identified by gene content rather than by cell-surface protein marker.

CHEMISTRY

Going for gold

Nano Lett. doi:10.1021/nl902186v (2009)

To increase conductance in miniaturized circuits, just add gold.

Paul Alivisatos at the University of California, Berkeley, and his colleagues were looking to see how the interface between a semiconductor nanorod and a metal would affect conductance. So they immersed the 40-nanometre-long cadmium selenide rods in a solution containing gold. This capped the rod tips with gold directly, and avoided the formation of a gold–semiconductor alloy, or a surfactant layer on the nanorod tip — both consequences of other rod-making procedures.

The procedure placed gold atoms on the tips of the rods and decreased the barrier to conductance — known as a Schottky barrier — giving them 100,000 times improved behaviour.

EVOLUTION AND DEVELOPMENT

Genes in the mirror

Curr. Biol. doi:10.1016/j.cub.2009.07.065 (2009)

Gene duplications may serve as the raw material for evolutionary changes in physical traits. Now researchers have found an example of how this might work in mutant and domesticated fish.

Nicolas Rohner and Matthew Harris of the Max Planck Institute for Developmental Biology in Tübingen, Germany, and their co-workers created a zebrafish (*Danio rerio*, pictured in upper panel) that is mostly scaleless. They found that the mutation responsible is in the gene *fgfr1*, which is required for embryonic development. Scouring the fish's genome, the authors found a previously uncharacterized copy of the gene. The two compensate for one another during embryonic growth but control different traits in adulthood.

Mutations in the same gene cause the characteristic scale pattern seen in the domesticated mirror carp (*Cyprinus carpio*, pictured in lower panel), providing a real-world example of how gene duplication can form the basis of new traits.

N. ROHNER

M. BERCSÉNYI

NEUROSCIENCE

Fear net

Science **325,** 1258–1261 (2009)

When adult rats are trained to associate a sound with an electric shock, they will often fear that sound for a lifetime. Young rats, however, can erase the fear memory when it is no longer relevant.

Andreas Lüthi, Cyril Herry and their colleagues at the Friedrich Miescher Institute for Biomedical Research in Basel, Switzerland, found that the reason for the shift may be the development of the perineuronal net, an extracellular protein lattice that surrounds a subset of neurons as rats mature. When the authors used an enzyme to dissolve the perineuronal net in the amygdala, a crucial brain region for forming fear memories, they found that adult rats could wipe away the memory of the shock as if they were young.

GENETICS

Why Y knots

Cell **138,** 855–869 (2009)

Theory predicts the chipping away of the male Y chromosome owing to the fact that, for the most part, it has no recombination partner during meiosis, the sexual form of cell division. But male-specific genes on the Y persist, protected in part by palindromic DNA repeats that are maintained through recombination events between each other. These repeats, say David Page of the Whitehead Institute for Biomedical Research in Cambridge, Massachusetts, and his collaborators, can also be Y's Achilles' heel.

The researchers propose a mechanism by which a crossover event after chromosomal replication links the two copies of the chromosome together at a palindrome, creating a larger, abnormal chromosome. These 'isodicentric' Y chromosomes are implicated in sex reversal, Turner's syndrome and male infertility. In a study of samples from 2,380 patients with suspected Y-chromosome defects, the authors identified 51 that apparently formed by this mechanism.

JOURNAL CLUB

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A biologist is gratified to find reconciliation for a conflicted receptor.

When giving talks on the involvement of the Eph family of receptor tyrosine kinases in cancer, I sometimes include a slide of the two-faced Roman god, Janus, to signify the dichotomies of Eph function in cancer cells. Most proteins have a clear-cut function. Some 'moonlighting' proteins carry out two unrelated functions. It is, however, rare for a protein to toggle between opposing activities. The Eph receptors are proving to be such outliers.

High expression of Eph receptors has been correlated with a poor cancer prognosis, but so has Eph silencing. Accordingly, there is good evidence that the Eph receptors can promote as well as inhibit tumour development. In a reconciliation reminiscent of Hegelian synthesis, a recent paper begins to explain how the EphA2 receptor can both promote and inhibit cancer cells' migratory and invasive abilities.

EphA2 activation by ephrin ligands seems to be minimal in most types of cancer cell. Hui Miao and Bingcheng Wang of Case Western Reserve University in Cleveland, Ohio, and their co-workers have shown that the protein Akt — which can be powerfully cancer-promoting — hijacks EphA2 by phosphorylating one of its serine residues, enabling its pro-metastatic activities (H. Miao *et al. Cancer Cell* **16,** 9–20; 2009).

Remarkably, binding by the ephrin-A1 ligand erases this phosphorylation and transforms EphA2 into an anti-invasive molecule.

These findings lead to the counterintuitive proposition that we should encourage rather than inhibit EphA2's ligand-dependent function. It will be interesting to see whether analogous switches convert other Eph receptors between malignant and benign phenotypes.

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