seeking to light creatures up from the inside, no natural proteins fluoresce very well at these wavelengths. Dmitriy Chudakov of the Shemiakin-Ovchinnikov Insititute of Bioorganic Chemistry in Moscow, Russia, and his colleagues created and tested more than 100,000 mutant forms of a red sea anemone protein to find one with a longer-wavelength glow. This protein, dubbed Katushka, was brighter than two existing far-red fluorescent proteins in tests with transgenic frogs. The researchers have also made a monomeric version of the protein, mKate, to use as a tag on other proteins.

PHYSIOLOGY

An eye on the time

Cell **130,** 730–741 (2007)

Switching off the eye's circadian 'clock' disrupts visual processing in mice, researchers have found. Many organs of the body have such clocks, but their function has been unclear.

Charles Weitz at Harvard Medical School and his colleagues deleted a circadian control gene, *Bmal1*, in the retina of mice. They noted that this disturbed the normal night and day cycle of retinal electrical activity.

By contrast, disrupting the central circadian clock in the brains of wild-type mice did not significantly affect the rhythm of the retinal impulses. This suggests the retinal circadian clock has an autonomous role in visual processing. Extensive changes in light-dependent gene expression are thought to be associated with the effect.

OPTICS

Stuck in a loop

Phys. Rev. A **76,** 023816 (2007)

Light ricocheting around inside a silica sphere like sound in a whispering gallery can be brought to a stop, researchers have shown.

Anatoliy Savchenkov and his colleagues at the Jet Propulsion Laboratory in Pasadena, California, calculated that a series of pulses of light entering a 'whispering-gallery-mode' resonator — a device that accepts only certain wavelengths of light — can end up with a zero 'group velocity'. This theoretical prediction runs against general expectations, but the team confirmed it through experiments.

Researchers have proposed using other systems that slow light for information storage. Savchenkov says it should be possible to build a resonator memory, although the stopped light in the resonator as configured in these experiments could hold no information.

STEM CELLS

Hope for the broken-hearted

Nature Biotechnol. doi:10.1038/nbt1327 (2007)

Poets tell us that, once broken, the heart does not readily heal. But progress in repairing damaged rat hearts with human embryonic stem cells raises hopes that such treatments may one day work for humans.

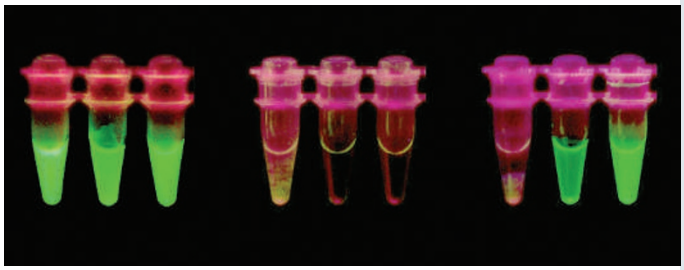
Charles Murry at the University of Washington in Seattle and his colleagues report that human embryonic stem cells treated with two proteins, activin A and bone morphogenic protein 4, develop into heart cells more efficiently than those treated with current methods. They also concocted a 'prosurvival cocktail' that blocked potential cell-death pathways, improving the survival of stem-cell derived heart cells when they were transplanted into rats. In rats that had had a heart attack, these cells replaced dead tissue, and prevented deterioration of heart function.

CHEMICAL BIOLOGY

Unscrambling the egg

J. Am. Chem. Soc. **129,** 10110–10112 (2007)

Anyone who has boiled an egg will have noticed the solidification that occurs as heat causes proteins to unfold then aggregate. Such aggregation can be a problem for the therapeutic use of proteins, so scientists are interested in schemes that might prevent it.



AM. CHEM. SOC.

David Liu and his colleagues at Harvard University, have found a way to make various proteins resistant to aggregation. They 'supercharge' the protein surfaces by substituting amino acids that appear on the outside surface of the folded protein with amino acids that can be ionized. Surprisingly, this does not seem to interfere with the protein's structure or function — but it keeps the molecules soluble after boiling, whereas unmutated forms aggregate.

The picture (above) shows solutions of the biochemical marker green fluorescent protein before (left) and immediately after boiling (centre), and after cooling (right). The leftmost vial in each set contains the standard protein, the other two contain supercharged forms.

JOURNAL CLUB

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To an epigeneticist, cancer is encrypted in genes and their packaging.

Early in my career I had the good fortune to study epigenetics in a lab focused on the molecular genetics of cancer. At the time, geneticists typically thought that in cancer, epigenetic changes — which affect regulation of the genome but not the genome's sequence — were epiphenomena less worthy of study.

This might have made the experience akin to being a Republican mayoral candidate in left-leaning San Francisco; instead it was positively transforming.

As my own research group took shape, I began to integrate genetic and epigenetic theories of malignant transformation. Now, hereditary human cancers and genetically engineered mice once held up as evidence for genetic models also provide evidence for epigenetic models, and we study the interactions of the two mechanisms.

In this light, a recent paper (G. G. Wang *et al. Nature Cell Biol.* **9,** 804–812; 2007) captured my attention because it dissects how one genetic change leads to epigenetic changes that ultimately cause leukaemia.

The work focuses on an abnormal fusion protein — produced after part of one gene fused, or translocated, with part of another — and narrows down its cancer-causing properties to a particular region of the protein. This region mediates an epigenetic change: it adds a methyl group to one amino acid of a histone, part of a gene's packaging in the nucleus.

The team found that the fusion protein misdirects its methylation to the histones that package *HoxA* genes, triggering further miscoding of the histones. This activates the genes, which promote self-renewal of blood-cell precursors, contributing to leukaemia.

I wonder if the interactions could be traced back even further. Given the role of epigenetics in stabilizing chromosomes; might it have been epigenetic miscoding that made the gene susceptible to translocation in the first place?

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