■ CANCER

Stem cell killer

Researchers have devised a way to screen for compounds that kill cancer stem cells, cells thought to seed and propagate a tumor that resist many conventional drugs.

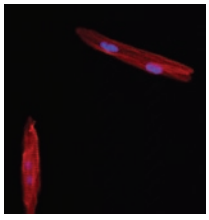
Such cells have been tough to cultivate in culture. To get around this problem, Piyush Gupta *et al.* introduced a gene into breast cancer cells that coaxed them to morph from epithelial cells into mesenchymal cells (*Cell* **138**, 645–659). These cells have similar molecular properties as cancer stem cells.

After screening a library of compounds, they homed in on one, salinomycin, an antibiotic commonly fed to livestock. Salinomycin could, somehow, kill cells thought to be stem cells in breast cancer cell lines. Such an approach has the potential to lead to drugs that can access stem cells *in vivo* and augment current therapies to knock out a tumor at its source. —*CS*

■ CARDIOVASCULAR DISEASE

Divide and repair

Previous attempts to regenerate the heart with cell therapy have generally used stem or progenitor cell types. Now, a report in *Cell* raises the profile of a different approach, exploiting the ability of differentiated cardiomyocytes to reenter the cell cycle and proliferate (*Cell* **138**, 257–270).



Kevin Bersell

Two nuclei from one, using ErbB4.

Kevin Bersell *et al.* found that the growth factor neuregulin-1 (NRG1), previously known to be needed for fetal heart development, can promote cardiomyocyte cell division. Cardiomyocytes can contain either one or two nuclei, but only the mononucleated variety could be stimulated to divide. Increasing the expression level of ErbB4, a receptor for NRG1, increased cardiomyocyte proliferation in mice, and removing ErbB4 had the opposite effect. Injection of NRG1 into young mice one week after myocardial infarction improved heart function, associated with a substantial increase in cardiomyocyte cell number.

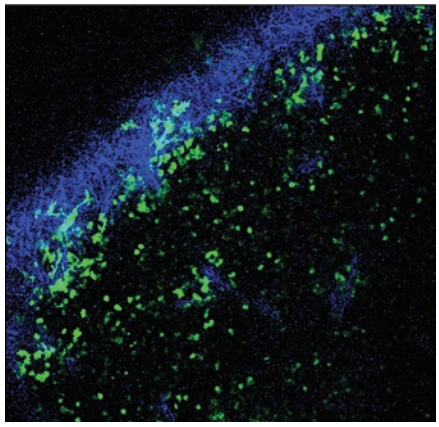
Although cardiomyocytes have typically been thought to be incapable of proliferation, this work adds to a growing body of evidence that, with the right stimulus, differentiated cardiomyocytes can indeed proliferate, helping to repair the heart after injury. —*MB*

■ IMMUNOLOGY

Storage in the spleen

Monocytes—cells of the innate immune system—hang out in the spleen before being recruited to damaged tissue, report Filip Swirski, Matthias Nahrendorf *et al.* (*Science* **325**, 612–616).

These cells are a heterogeneous population that help defend against infection and help repair damaged tissue. They arise from the bone marrow and, until now, were thought to circulate in the blood stream until needed at sites of tissue damage or infection.



AAAS

Monocytes (green) in the spleen (collagen in blue).

The authors began with a curious observation: they found that damaged mouse heart accumulates more monocytes than are present in the circulation. To identify the reservoir of these cells the authors screened a variety of tissues and found that only the spleen harbored large numbers of monocytes. To prove the monocytes came from the spleen and not the bone marrow, the authors induced myocardial infarction in splenectomized mice, or mice lacking the receptor CCR2, which is required for monocytes to exit the bone marrow. The number of blood monocytes increased after infarction in CCR2-deficient mice but not in the mice lacking a spleen.

Mammals can survive in the absence of a spleen, as most of its jobs, such as red blood cell synthesis, can be taken over by other organs. But the new findings show the spleen has more functions than previously supposed. —*CT*

Written by Michael Basson, Kevin Da Silva, Katherine Gora, Roberta Kwok, Charlotte Schubert and Clare Thomas

New from NPG

Initiation of myoblast to brown fat switch by a PRDM16-C/EBP-β transcriptional complex.
Kajimura, S. *et al. Nature* doi:10.1038/nature08262 (29 July).
Researchers turn myoblast precursors into brown fat, getting closer to harnessing this fuel-burning cell to counteract obesity and type 2 diabetes.

Gain-of-function of mutated C-CBL tumour suppressor in myeloid neoplasms.
Sanada, M. *et al. Nature* **460**, 904–908.
Loss of heterozygosity uncovers a gain-of-function mutation that promotes tumorigenesis.

HIV-1 evades virus-specific IgG2 and IgA responses by targeting systemic and intestinal B cells via long-range intercellular conduits.
Xu, W. *et al. Nat. Immunol.* doi:10.1038/ni.1753 (2 August).
In response to Nef, an HIV-encoded molecule that suppresses the immune system, macrophages form actin-propelled conduits to B cells. This conduit delivers Nef to the cells, aiding in immune suppression.

Deaf1 isoforms control the expression of genes encoding peripheral tissue antigens in the pancreatic lymph nodes during type 1 diabetes.
Yip, L. *et al. Nat. Immunol.* **10**, 1026–1033.
This study examines how peripheral immune tolerance breaks down during type 1 diabetes.

Single-molecule sequencing of an individual human genome.
Pushkarev, D., Neff, N.F. & Quake, S.R. *Nat. Biotechnol.* doi:10.1038/nbt.1561 (10 August).
Sequence emerges from single molecules, bypassing need for cumbersome cloning and amplification steps.