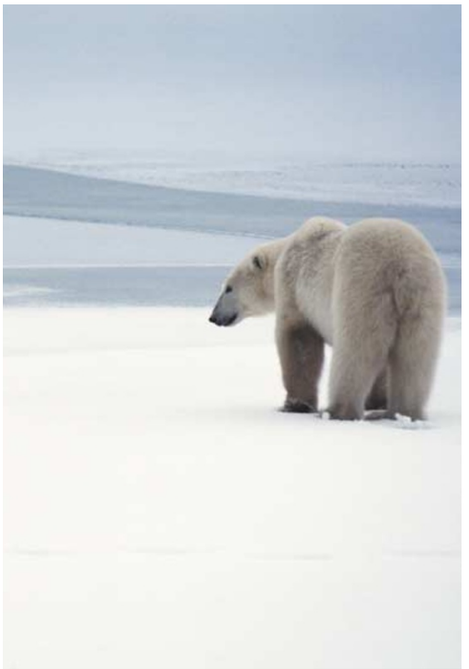

in a DivIVA mutant strain is impaired, a MinD DivIVA double-mutant strain was used. RacA–GFP failed to localize to the extreme poles, whereas localization in a MinD mutant was similar to the wild-type strain, which indicates that RacA localization is indeed dependent on DivIVA.

Chromatin immunoprecipitation and subsequent PCR experiments confirmed that RacA colocalizes with the entire nucleoid, and also revealed preferential binding sites for RacA in the replication origin region.

Losick and colleagues have therefore proposed a model in which RacA is a kinetochore-like protein that binds preferentially near the replication origin and anchors the chromosome to the cell pole by binding — directly or indirectly — to DivIVA. In addition, nonspecific binding of RacA throughout the nucleoid allows remodelling of the chromosome into an axial filament structure. A possible role for RacA in polar division requires further investigation.

Arianne Heinrichs

References and links

ORIGINAL RESEARCH PAPER Ben-Yehuda, S. *et al.* RacA, a bacterial protein that anchors chromosomes to the cell poles. *Science Express* 19 December 2002 (DOI: 10.1126/science.2079914)

WEB SITE

Richard Losick's laboratory: http://mcb.harvard.edu/losick/

influences the inside–out transducing function of syndecan-2 in the ectoderm to enable it to act cell non-autonomously to influence the migrating mesodermal cells. However, there seems to be no shortage of ideas.

Katrin Bussell

References and links

ORIGINAL RESEARCH PAPER Kramer, K. L., Barnette, J. E. & Yost, H. J. PKCγ regulates syndecan-2 inside–out signaling during *Xenopus* left–right development. *Cell* **111**, 981–990 (2002)

FURTHER READING Essner, J.J. *et al.* Conserved function for embryonic nodal cilia. *Nature* **418**, 37–38 (2002)

WEB SITE

Joseph Yost's laboratory: http://www.hci.utah.edu/groups/yost/

IN BRIEF

DEVELOPMENT

Heart regeneration in zebrafish.

Poss, K. D. *et al*. *Science* **298**, 2188–2190 (2002)

In most vertebrates, cardiac injury leads to scar formation. By contrast, zebrafish can regenerate cardiac tissue following mechanical injury, as is now shown by Mark Keating and colleagues. Cardiomyocyte proliferation occurs at the leading epicardial edge, and regeneration is complete within 2 months of 20% ventricular resection. However, zebrafish with a mutation in the Mps1 mitotic checkpoint kinase do not regenerate and form scar tissue.

PROTEIN TRANSLOCATION

Molecular chaperones Hsp90 and Hsp70 deliver preproteins to the mitochondrial import receptor Tom70.

Young, J. C. *et al*. *Cell* **112**, 1–20 (2003)

The delivery of preproteins from the cytosol to mitochondria is poorly understood. Young *et al.* now report that, in mammals, the cytosolic chaperones Hsp90 and Hsp70 dock onto the import receptor Tom70 at the outer mitochondrial membrane. This interaction is essential for targeting a subset of preproteins to the receptor for subsequent import. However, in yeast, only Hsp70 docking is needed for effective preprotein delivery.

BIOENERGETICS

SRC-1 and TIF2 control energy balance between white and brown adipose tissues.

Piccard, F. *et al*. *Cell* **111**, 931–941 (2002)

Piccard *et al.* found that two members of the p160 coregulator family — TIF2 and SRC-1 — have a function in the energy homeostasis of white and brown adipose tissues. *TIF2*$^{-/-}$ mice are protected against excessive fat accumulation and have increased insulin sensitivity. By contrast, *SRC1*$^{-/-}$ mice are prone to obesity due to reduced energy expenditure. These phenotypes are caused by changes in the expression ratio of TIF2 and SRC-1 leading to an altered composition of coregulator complexes, which, in turn, affects the transcriptional control of fat storage and thermogenesis.

APOPTOSIS

c-MYC apoptotic function is mediated by NRF-1 target genes.

Morrish, F. *et al*. *Genes Dev*. **17**, 240–255 (2003)

Although earlier studies indicated a link with the mitochondrial apoptotic signalling pathway, the precise mechanism by which c-Myc induces apoptosis has remained elusive. Morrish *et al.* now show that c-Myc can stimulate target genes of the nuclear respiratory factor (NRF)-1, a transcription factor for several mitochondrial-related genes, including *cytochrome c*. Under conditions that trigger c-Myc-induced apoptosis, overexpression of NRF-1 sensitizes cells to apoptosis. Also, a dominant-negative NRF-1 mutant inhibits c-Myc-induced apoptosis but not c-Myc-induced proliferation.