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IN BRIEF

DEVELOPMENT

Histone arginine methylation regulates pluripotency in the early mouse embryo.

Torres-Padilla, M. -E. *et al. Nature* **445**, 214–218 (2007)

Cells in the mouse embryo differ in their developmental fates as early as at the four-cell stage. So how might cell fate be regulated at this early stage? Torres-Padilla and colleagues show that methylation of histone H3 on arginine residues correlates with the development of cells into the inner cell mass (ICM). By contrast, cells that form the trophectoderm show minimal methylation at selected arginine residues. Overexpression of the arginine methyltransferase enzyme CARM1 in one cell of a two-cell blastomere resulted in the majority of the CARM1-cell progeny forming the ICM and an increase in the levels of Nanog and SOX2, two transcription factors that function during early ICM development. Histone arginine methylation is the earliest epigenetic mark involved in the control of cell-fate decisions that has been identified so far.

PROTEIN DEGRADATION

Ubiquitin chains are remodelled at the proteasome by opposing ubiquitin ligase and deubiquitinating activities.

Crosas, B. *et al. Cell* **127**, 1401–1403 (2006)

The proteasome is a multisubunit complex that recognizes and degrades ubiquitylated proteins. The group of Finley reports that the ubiquitin ligase Hul5, which is associated with the proteasome, can extend ubiquitin chains on protein substrates and enhance their degradation. *Hul5*-mutant yeast show defect in the degradation of multiple proteins, and the ubiquitin chains deposited by Hul5 can be efficiently removed by the proteasome-bound deubiquitylating enzyme Ubg6. The authors show that the association of Hul5 with the proteasome is drastically reduced by Ubg6, leading them to propose that the two enzymes are localized with close proximity on the proteasome. Together, the chain-extending and chain-trimming activities of Hul5 and Ubg6 might be a final regulatory checkpoint before a protein is committed to degradation by the proteasome.

DEVELOPMENT

β cells occur naturally in extrahepatic bile ducts of mice.

Dutton, J. R. *et al. J. Cell Sci.* **120**, 239–245 (2007)

Dutton *et al.* report, for the first time in mammals, the existence of insulin-secreting β cells outside the pancreas. The authors show that the bile duct epithelium contains small populations of β cells and other cell types that are usually found in the endocrine pancreas. By genetic lineage labelling, the authors demonstrate that these β cells are derived from cells that originate in the liver, rather than the pancreas, during embryonic development. Given that some primitive vertebrates derive all their β cells from the biliary duct, this β-cell population that has been identified in mice might be an evolutionary remnant. This finding has implications for researchers that are trying to reprogramme cellular tissues into β cells that can be transplanted into patients with diabetes. Although it remains unknown whether β cells reside in the bile duct in humans, the bile duct epithelium is now a candidate tissue from which β cells might be generated.