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10.1038/nrm2380

IN BRIEF

CYTOSKELETON

The structural basis of actin filament branching by the Arp2/3 complex.

Rouiller, I. *et al. J. Cell Biol.* 3 March 2008 (doi:10.1083/jcb200709092)

The actin-related protein-2/3 (ARP2/3) complex initiates actin filament branching at an angle on the sides of pre-existing mother filaments. The authors used electron tomography to reconstruct the branch junction and visualize how the ARP2/3 complex interacts with the mother filament. Branch formation causes the ARP2 and ARP3 subunits to rearrange next to each other to form the first two subunits of the daughter filament. The mother filament also undergoes conformational changes, which help to increase the stability of the branch.

STEM CELLS

A skin microRNA promotes differentiation by repressing 'stemness'.

Yi, R. *et al. Nature* 2 March 2008 (doi:10.1038/nature06642)

The authors showed that by functioning as a switch between proliferation and differentiation, the microRNA miR-203 defines a molecular boundary between basal progenitors and suprabasal cells in epidermal tissue. miR-203 is induced in the suprabasal cells of stratified, differentiated epithelial tissues, but not in the multipotent progenitor cells of single-layered epidermis. Premature activation of miR-203 in the epidermis restricts its proliferative potential, whereas inhibition of miR-203 results in increased epidermal proliferation. miR-203 directly represses expression of the *p63* gene, which encodes an essential regulator of stem-cell maintenance in stratified epithelial tissues.

RNA SILENCING

MicroRNAs control *de novo* DNA methylation through regulation of transcriptional repressors in mouse embryonic stem cells.

Sinkkonen, L. *et al. Nature Struct. Mol. Biol.* 2 March 2008 (doi:10.1038/nsmb.1391)

A mammalian microRNA cluster controls DNA methylation and telomere recombination via Rbl2-dependent regulation of DNA methyltransferases.

Benetti, R. *et al. Nature Struct. Mol. Biol.* 2 March 2008 (doi:10.1038/nsmb.1399)

What are the functional consequences of deficiency of the RNA silencing enzyme Dicer? Sinkkonen *et al.* showed that *Dicer1* $^{-/-}$ mouse embryonic stem cells fail to undergo *de novo* DNA methylation, which correlates with the decreased expression of certain DNA methyltransferases. The responsible transcriptional repressor, retinoblastoma-like protein-2 (RBL2), is a probable target of the miR-280 cluster, which is downregulated in Dicer1-deficient cells. Benetti *et al.* reported the same findings in mouse myoblast cells and, in addition, showed that Dicer deficiency does not lead to a decrease in the levels of repressive histone trimethylation marks. This finding suggests that Dicer and, therefore, Dicer-dependent small RNAs are not required for the establishment of regions of silenced chromatin in mice. This is in contrast to the well-established role for Dicer-dependent small RNAs in heterochromatin formation and function in fission yeast and the fly.