IN BRIEF

EPIGENETICS

Methylation in paternal inheritance

During the final stages of sperm formation, most histones are replaced by sperm-specific protamine proteins to enable DNA compaction in sperm nuclei. The function of the retained nucleosomes in transgenerational inheritance is largely unknown. Siklenka *et al.* reduced the dimethylation of histone H3 Lys 4 (H3K4me2) in mouse sperm by overexpressing the human Lys demethylase KDM1A (also known as LSD1) specifically in the male germ line. This led to H3K4me2 loss in many developmental genes. The offspring of heterozygous transgenic males showed severe developmental defects, which were transmitted paternally through three generations, even when *KDM1A* was not expressed in the offspring germ line. No changes in DNA methylation were observed at CpG islands, whereas RNA profiles were altered in the sperm of transgenic males and their offspring, suggesting an important role for sperm histone methylation in transgenerational inheritance.

ORIGINAL RESEARCH PAPER Siklenka *et al.* Disruption of histone methylation in developing sperm impairs offspring health transgenerationally. *Science* http://dx.doi.org/10.1126/science.aab2006 (2015)

CELL ADHESION

SUMO controls a tug of war at junctions

Adherens junctions are complexes that comprise E-cadherin molecules, which interact with each other on neighbouring cells, and with the underlying actin cytoskeleton at the apical surface of epithelia, to establish cell–cell contacts. These contacts are necessary for the maintenance of epithelial integrity but need to be disassembled to allow tissue remodelling and cell migration. Tsur *et al.* now show that the balance between adherens junction assembly and disassembly is regulated by the small ubiquitin-like modifier (SUMO) protein, which can be added to or removed from E-cadherin molecules. Specifically, they reveal that removal of the SUMO mark, mediated by a defined protease, is necessary for proper apical localization of E-cadherin and its interaction with actin, thus representing a key step in junctional assembly. Altogether, the authors uncover SUMO as an important modulator of adherens junctions. They suggest that SUMO molecules are constantly added to and removed from E-cadherin, thereby providing a mechanism to regulate adherens junction dynamics, which are crucial for morphogenesis and tissue homeostasis.

ORIGINAL RESEARCH PAPER Tsur, A. *et al.* ULP-2 SUMO protease regulates E-cadherin recruitment to adherens junctions. *Dev. Cell* http://dx.doi.org/10.1016/j.devcel.2015.08.019 (2015)