

IN BRIEF

 CHROMOSOMES**Finding your pair**

The pairing and recombination of homologous chromosomes is crucial for their reductional segregation during meiosis I, but until now it has been unclear how chromosomes recognize their homologous partners. Ding *et al.* observed that fluorescently tagged *sme2* loci, located on *Schizosaccharomyces pombe* chromosome 2, paired frequently in early meiotic prophase. When the *sme2* locus was translocated to another region, robust pairing was seen at this ectopic site, indicating that *sme2* is sufficient to induce robust homologous pairing. Furthermore, when two copies of *sme2* were present on non-homologous chromosomes, the two loci associated transiently, suggesting that *sme2* loci can recognize each other. Importantly, *sme2* non-coding RNA transcripts, transcribed from both homologous chromosomes, accumulated at their respective gene loci and were found to be essential for pairing. Whether other pairing sites also exist on chromosome 2 remains to be determined.

ORIGINAL RESEARCH PAPER Ding, D.-Q. *et al.* Meiosis-specific noncoding RNA mediates robust pairing of homologous chromosomes in meiosis. *Science* **336**, 732–736 (2012)

 CELL ADHESION**Cell–cell contacts with talin**

Talin has a key role in integrin-dependent adhesion of cells to the extracellular matrix, and Kashina and colleagues now report that talin also mediates cell–cell adhesion. A 70 kDa carboxy-terminal fragment (termed VAD) of talin was seen in several cell types and localized at cadherin-containing cell–cell adhesion sites. This fragment was generated through cleavage of talin by the protease calpain 2, and cleavage was dependent on arginylation of VAD. Importantly, arginylation-deficient mutant cells that lacked the VAD fragment formed fewer and smaller cell–cell adhesions, indicating a role for VAD in cell–cell contacts. Furthermore, arginylation was integral in the regulation of VAD function, as arginylated VAD was more efficient in forming cell–cell contacts than its non-arginylated counterpart. Thus, this work uncovers a new role of talin in cadherin-dependent cell–cell adhesion and highlights arginylation as an important post-translational modification.

ORIGINAL RESEARCH PAPER Zhang, F. *et al.* Arginylation-dependent regulation of a proteolytic product of talin is essential for cell–cell adhesion. *J. Cell Biol.* 4 Jun 2012 (doi:10.1083/jcb.201112129)

 CELL SIGNALLING**Breaking down cilia**

The disassembly of non-motile cilia (the antenna-like structures that transduce extracellular signals) prior to mitosis has been shown to be mediated by the interaction of the scaffold protein HEF1 with Aurora A kinase, but how these proteins are activated had remained unclear. Here, Lee *et al.* reveal key roles for Polo-like kinase 1 (PLK1) and the WNT signalling mediator Dishevelled 2 upstream of HEF1 and Aurora A. The authors observed that, following activation by the non-canonical WNT ligand WNT5A, casein kinase 1 δ (CK1 δ) or CK1 ϵ phosphorylated Dishevelled 2 at Ser143 and Thr224, inducing the formation of a Dishevelled 2–PLK1 complex. Further analysis showed that the Dishevelled 2–PLK1 complex induced by CK1 ϵ is important for the stabilization of HEF1 (in a manner dependent on the kinase activity of PLK1) and thus for the interaction of HEF1 with Aurora A, ultimately leading to cilium disassembly.

ORIGINAL RESEARCH PAPER Lee, K. H. *et al.* Identification of a novel WNT5A–CK1 ϵ –DVL2–PLK1-mediated primary cilia disassembly pathway. *EMBO J.* 18 May 2012 (doi:10.1038/emboj.2012.144)