IN BRIEF

CELL ADHESION

Sticking three cells together

Luschnig and colleagues have identified a new component of Drosophila melanogaster tricellular junctions (TCJs), Anakonda (Aka), which is specifically required for TCJ assembly. TCIs connect epithelial cells where the corners of three adjacent cells meet and are important for cytoskeletal organization and epithelial barrier function. aka mutants had excessively long and tortuous tracheal tubes and were disrupted in cell-cell adhesion and epithelial barrier functions. Aka is a large transmembrane protein with an unusual tripartite extracellular domain that functions early in TCI formation by recruiting or maintaining Gliotactin, which is the only other characterized TCJ component in invertebrates. The cytoplasmic domain of Aka was dispensable for TCJ assembly, and TCJs assembled correctly only if Aka was present in all three cells of a three-cell vertex. These observations, combined with computational modelling, indicate that TCJ assembly is driven by Aka-mediated extracellular interactions and the geometry of tricellular vertices.

ORIGINAL RESEARCH PAPER Byri, S. et al. The triple-repeat protein Anakonda controls epithelial tricellular junction formation in Drosophila. Dev. Cell **33**, 535–548 (2015)

TECHNIQUES

microRNA switches to isolate specific cells

For many cell types, no specific cell-surface markers that would facilitate their isolation have been identified. In this study, Miki *et al.* describe a method to isolate specific cell types on the basis of their endogenous microRNA (miRNA) activity. The authors designed 'miRNA switches', which are synthetic mRNAs that encode fluorescent proteins and that contain a specific miRNA target site in their 5' untranslated region. Cells expressing the corresponding endogenous miRNA can be distinguished from other cells as they induce the translational repression of the reporter. This method enabled the purification of specific cell types differentiated from human pluripotent stem cells, such as cardiomyocytes, hepatocytes, endothelial cells and insulin-producing cells.

ORIGINAL RESEARCH PAPER Miki, K. et al. Efficient detection and purification of cell populations using synthetic microRNA switches. *Cell Stem Cell* **16**, 699–711 (2015)

DNA DAMAGE RESPONSE

Damage by Helicobacter pylori

Infection with the gastric bacterium Helicobacter pylori can cause chromosomal instability (CIN) and promote tumorigenesis. Koeppel et al. compared the DNA damage response (DDR) following H. pylori infection with that induced by other genotoxic treatments in human gastric cells and observed reduced activation of the DDR components ATR and the MRN complex, but not of 53BP1, following infection. Furthermore, the expression of 58 DDR genes was downregulated, indicating that *H. pylori* can systematically reduce DNA damage repair capacities. Measuring genome-wide localization of the DDR factor yH2AX revealed that by 18 hours following infection, damage induced by ionizing radiation shifted from being randomly localized to being localized to genic regions, especially to actively transcribed genes, and to telomere-proximal regions. This is similar to CIN patterns observed in H. pylori-associated gastric cancers.

ORIGINAL RESEARCH PAPER Koeppel, M. et al. Helicobacter pylori infection causes characteristic DNA damage patterns in human cells. Cell Rep. <u>http://dx.doi.org/10.1016/j.celrep.2015.05.030</u> (2015)