Pygo2 opens chromatin and cycles cells

Without Pygo2 (right) mammary glands have fewer cycling progenitor cells (brown).

By spreading an active chromatin state, Pygo2 prompts the proliferation of mammalian gland progenitor cells, report Gu et al.

The fly version of Pygo2, Pygopus, is essential for Wg signaling. But the relationship between mammalian Wg (Wnt) and Pygo2 is less clear. Pygo2 is necessary for the development of a number of tissues, but in the two best studied—eye and testis—it has no need for Wnt.

Gu et al. looked at the relationship between Wnt and Pygo2 in mammary gland epithelial cells, where both proteins have been linked with cancer. Pygo2 was expressed in mammary progenitor cells in the embryo and adult mouse, where it seemed to specifically regulate proliferation.

Heart saves muscle

A heart muscle protein can replace its missing skeletal muscle counterpart to give mice with myopathy a long and active life, show Nowak et al.

The contraction machinery protein, actin, exists in different forms in the adult heart and skeletal muscles. The heart form, ACTC, is also the dominant form in skeletal muscle of the fetus. But during development, the skeletal form, ACTA1, increases in production and by birth has taken over. It is not clear why the switch occurs, or why it doesn’t occur in the heart, but it happens in every higher vertebrate and, for that reason, has been considered vitally important.

Mutations to the ACTA1 gene cause a rare but serious myopathy. Most patients die within the first year of life and some are born almost completely paralyzed. Mice lacking ACTA1 die nine days after birth.

Dynamics of staying put

Cadherin clusters (green) form between microvilli (inset) before teaming up with Bazooka clusters at lateral membranes.

Without cell–cell connections our bodies would fall apart. McGill et al. have now delved into the dynamics of connection construction.

The connections are called adherens junctions. Within each cell these junctions are built, not by assembling proteins at single sites, but by bringing two different protein complexes together, the team now shows. One of the complexes, Bazooka clusters, remains steadfast at the cell cortex and catches the other complex, the cadherin–catenin clusters, as they flow along in the membrane.

To determine these dynamics, the team followed fluorescently tagged versions of the complexes in fly embryos at a stage called cellularization—when one giant multinucleated cell becomes an epithelial layer of mononucleated cells.

Bazooka clusters formed at the contacts between these cells. Meanwhile, cadherin–catenin clusters first formed between microvilli structures on the apical surface. They then moved down to the cell–cell contacts, where the Bazooka clusters were waiting.

In between microvilli might seem like a strange place to form complexes involved in cell–cell contact, but senior author Tony Harris suggests that the movement of the microvilli membranes might help accumulate the cadherin and catenin into clusters. Also, at the transition region between apical and lateral (cell–cell contact) membranes, microvilli can interlock. This could then produce clusters between neighboring cells enabling the cells to grab hold of each other.

McGill, M.A., et al. 2009. J. Cell Biol. doi:10.1083/jcb.200812146.