Apathway that clears up the remains of dead cells also removes the debris generated during cell division, Chai et al. report.

The midbody is a microtubule-rich structure that connects the two daughter cells at the end of mitosis. When cytokinesis is completed, the midbody is either shed into the extracellular space or retained by one of the daughter cells, potentially influencing its developmental fate. Chai et al. found that, in C. elegans, asymmetrically dividing Q neuroblasts discard their midbodies into their surroundings.

The Q cell midbodies were subsequently engulfed and degraded by a neighboring epithelial cell called hyp7, which also internalizes the corpses of apoptotic Q cells during development. Mutations in the genes that promote apoptotic cell engulfment blocked midbody clearance, indicating that hyp7 cells use the same pathway to internalize Q cell corpses and cytokeratin midbodies. Indeed, Chai et al. found that, just like apoptotic cells, Q cell midbodies expose phosphatidylserine on their outer surface and that blocking this lipid signal prevented the hyp7 cell from recognizing and engulfing the remnants of Q cell divisions.

Apoptotic engulfment genes also regulated midbody clearance in other C. elegans cell lineages. But senior author Guangshuo Ou now wants to study the function of the midbodies produced by worm epithelial stem cells, which are specifically retained by the daughter cell that remains undifferentiated.

How endothelial junctions get deep-Syx’d

Ngok et al. describe how two growth factors control the localization of a guanine nucleotide exchange factor (GEF) to exert opposing effects on vascular permeability.

Vascular endothelial growth factor (VEGF) increases blood vessel leakiness by disrupting the junctions between endothelial cells, whereas Angiopoietin-1 (Ang1) stabilizes intercellular adhesions to decrease vascular permeability. Intercellular junctions are regulated by Rho family GTPases and by polarity proteins like the Crumbs complex. Ngok et al. discovered that a GEF called Syx localizes to endothelial cell junctions by binding to the Crumbs complex member Mupp1. Syx stabilized intercellular adhesions by locally activating RhoA and its downstream effector, the formin Dia.

VEGF disrupted endothelial cell contacts by displacing Syx from intercellular junctions. The growth factor inhibited Syx’s association with Mupp1 by stimulating its phosphorylation by protein kinase D1 (PKD1). A non-phosphorylatable Syx mutant remained bound to Mupp1 at cell junctions and prevented VEGF from destabilizing cell–cell contacts. Ang1 also prevented VEGF from displacing Syx and disrupting intercellular adhesions, but Ang1 was unable to stabilize junctions in the absence of Syx.

Mice lacking Syx had leaky blood vessels, resulting in edemas and reduced heart function. Increased vascular permeability is associated with many human diseases, including strokes and tumor metastasis. The authors now want to investigate whether promoting Syx’s localization to endothelial cell junctions, by inhibiting PKD1 for example, could be used to prevent vascular leakiness.

Ngok, S.P., et al. 2012. J. Cell Biol. http://dx.doi.org/10.1083/jcb.201207009.