IN BRIEF

CELL SENESCENCE

Communicating with senescence

Senescent cells secrete molecules to communicate with surrounding cells. Biran *et al.* report that communication can also occur by intercellular protein transfer (IPT). When primary natural killer (NK) cells were co-cultured with senescent or normally growing human fibroblasts, IPT of a fluorescent protein was detected only in cultures with senescent cells and resulted in the activation of NK cell-mediated cytotoxicity. IPT required cell contact and was in part mediated by the formation of cytoplasmic bridges. Several transferred proteins were identified, including proteins related to actin cytoskeleton reorganization. IPT and NK cell cytotoxicity were indeed dependent on actin polymerization, which was mediated by the upregulation and activation of the GTPase CDC42 in senescent cells. IPT was also observed with epithelial cells and T cells, and *in vivo* in mice.

ORIGINAL RESEARCH PAPER Biran, A. *et al.* Senescent cells communicate via intercellular protein transfer. *Genes Dev.* http://www.genesdev.org/cgi/doi/10.1101/gad.259341.115 (2015)

CELL MIGRATION

Speed and persistence

Cell migration is essential for development and tissue repair, and is crucial for tumorigenesis. It is unclear whether a general rule can account for the diversity of cell migration patterns observed *in vivo* and *in vitro*. By analysing trajectories of cells migrating in live tissues and under various *in vitro* conditions, Maiuri *et al.* revealed a universal coupling between cell migration speed and cell persistence (the capacity to maintain the direction of motion). The authors developed a physical model to explain how faster cells move straighter. In this model, coupling between cell speed and persistence relies on cellular actin retrograde flows that transport polarity cues from the front to the rear of migrating cells. The model, which was validated experimentally by optogenetic modulation of actin retrograde flows, can predict cell trajectories and could thus be used for the study of processes such as metastasis.

ORIGINAL RESEARCH PAPER Maiuri, P. *et al.* Actin flows mediate a universal coupling between cell speed and cell persistence. *Cell* **161**, 374–386 (2015)

CELL DIVISION

Sorting ageing mitochondria

Stem cells can divide asymmetrically, generating a daughter stem cell and a cell that is committed for differentiation. Whether mammalian stem cells segregate organelles asymmetrically during cell division remains unclear. Katajisto *et al.* now show that daughter cells with stem cell properties inherit fewer 'old' mitochondria, indicating that asymmetric partitioning of mitochondria is important for the maintenance of stemness. Mitochondria were labelled (by marking the inner or outer mitochondrial membranes with photoactivatable GFP or SNAP-tag) in cultured human mammary epithelial cells that contain stem-like cells (SLCs). Old mitochondria (aged 10 hours or more) segregated asymmetrically in SLCs, as one daughter cell inherited ~5.6-fold more old mitochondria than the other. Daughter cells with more old mitochondria differentiated, whereas those with fewer old mitochondria remained SLCs. Asymmetric partitioning required the confinement of old mitochondria to the perinuclear region of the mother cell, and this was dependent on mitochondrial fission.

ORIGINAL RESEARCH PAPER Katajisto, P. *et al.* Asymmetric apportioning of aged mitochondria between daughter cells is required for stemness. *Science* http://dx.doi.org/10.1126/science.1260384 (2015)