IN BRIEF

CELL MIGRATION

Moving towards ECM with LKB1

Chan *et al.* investigated the mechanism by which loss of the Ser/Thr kinase LKB1 facilitates the invasion and metastasis of melanoma cells. Using scratch wound assays, which remove the extracellular matrix (ECM), they found that LKB1 null melanoma cells migrated into the wound, but null cells in which LKB1 was re-expressed (addback cells) aligned at the wound edge, suggesting that LKB1 might be involved in boundary sensing. Indeed, addback cells, but not LKB1 null cells, migrated towards ECM gradients (haptotaxis); both cell types migrated towards soluble chemical cues (chemotaxis). LKB1-mediated phosphorylation of MAP/microtubule affinity-regulating kinase (MARK) family members was essential for its role in haptotaxis, although the downstream targets of MARKs here are unidentified. Thus, the LKB1–MARK pathway is required for melanoma cell haptotaxis and its disruption may promote their invasion.

ORIGINAL RESEARCH PAPER Chan, K. T. *et al.* LKB1 loss in melanoma disrupts directional migration toward extracellular matrix cues. *J. Cell Biol.* doi: 10.1083/jcb.201404067 (2014)

CELL ADHESION

Basement membranes stick together

The basement membrane is a specialized sheet-like extracellular matrix (ECM) that surrounds most tissues. The mechanisms underlying adhesion between basement membranes, which is important for neighbouring tissue alignment, are unclear. Studying *Caenorhabditis elegans* uterine–vulval attachments, Morrissey *et al.* found that the ECM component hemicentin, and VAB-10A (plakin) and INA-1/PAT-3 (integrin), connect adjacent basement membranes. Genetic analyses, transmission electron microscopy, tissue shifting experiments and live-cell imaging revealed that hemicentin is secreted into the basement membrane by the anchor cell (a specialized uterine cell) prior to its invasion into the vulva, and forms punctae; these link the juxtaposed gonadal and ventral basement membranes to allow anchor cells to invade through them and establish uterine–vulval connections. INA-1/PAT-3 and VAB-10A were required to establish and stabilize hemicentin-containing adhesions, respectively.

ORIGINAL RESEARCH PAPER Morrissey, A. *et al.* B-LINK: A hemicentin, plakin, and integrin-dependent adhesion system that links tissues by connecting adjacent basement membranes. *Dev. Cell* doi.org/10.1016/j.devcel.2014.08.024 (2014)

PROTEIN DEGRADATION

Piecing together protein quality control

Ribosomes that cannot terminate translation pause and their 60S subunits remain stalled with peptidyl-tRNA-associated nascent chains. The listerin E3 ubiquitin protein ligase 1 (Ltn1) and its cofactors, which make up the large ribosomal subunit-associated quality control complex (RQC), then target these chains for proteasomal degradation. Lyumkis *et al.* determined the structure of the yeast RQC bound to stalled 60S ribosome subunits using single-particle cryoelectron microscopy. They revealed that the RQC subunit Tae2 recognizes, and interacts with, the exposed tRNA of the stalled 60S-associated peptidyl-tRNA, providing insight into how RQC selectively recognizes aberrant nascent chains. They also showed that the C terminus of Ltn1 binds to the 60S subunit near the nascent polypeptide exit tunnel, which positions the Ltn1 RING domain to ubiquitylate the stalled nascent chains. This study increases our understanding of how protein translation and degradation are co-regulated.

ORIGINAL RESEARCH PAPER Lyumkis, D. *et al.* Structural basis for translational surveillance by the large ribosomal subunit-associated protein quality control complex. *Proc. Natl Acad. Sci. USA* doi/10.1073/pnas.1413882111 (2014)