Nature Reviews Molecular Cell Biology | Published online16 March 2016

IN BRIEF

CELL ADHESION

Autophagy consumes integrin junctions

Focal adhesions are integrin-based complexes through which cells interact with the extracellular matrix, and their regulated assembly and disassembly underlies cell migration. How focal adhesion dynamics are regulated remains unclear. Kenific *et al.* demonstrate that autophagy is an important regulator of the turnover of these adhesive complexes. Inhibition of autophagy stabilized focal adhesions, reducing both their assembly and disassembly rates, and impaired migration of various cell types. The authors revealed that autophagosomes, which engulf cellular components during autophagy, co-localized with focal adhesions during their disassembly. Finally, they established that autophagy receptor NBR1 is involved in targeting focal adhesion components to the autophagosome. It will be interesting to study how autophagy cooperates with other mechanisms of focal adhesion remodelling.

ORIGINAL ARTICLE Kenific, C. M. *et al.* NBR1 enables autophagy-dependent focal adhesion turnover. *J. Cell Biol.* **212**, 577–590 (2016)

MECHANISMS OF DISEASE

Benefits of oxygen limitation

Mitochondrial dysfunction can be triggered by various genetic and environmental factors, leading to a broad range of diseases. To study pathways that might protect from mitochondrial damage, Jain *et al.* performed a CRISPR–Cas9-mediated knockout of ~18,000 genes, revealing that deletion of a suppressor of the cellular response to low oxygen (hypoxia) significantly improved growth of cells with dysfunctional mitochondria. This suggested that the hypoxic response protects cells from defects resulting from mitochondrial malfunction. Indeed, it was demonstrated both *in vitro* and *in vivo* in the zebrafish that raising the hypoxic response through suppression of the inhibitory pathway improved survival when mitochondrial respiration was perturbed. The authors also showed that exposure to a hypoxic environment prevented cellular malfunctioning, alleviated disease symptoms and extended the lifespan of mice suffering from mitochondrial dysfunction. The next exciting step would be to attempt to harness hypoxic response for clinical applications.

ORIGINAL ARTICLE Jain, I. H. *et al.* Hypoxia as a therapy for mitochondrial disease. *Science* http://dx.doi.org/10.1126/science.aad9642 (2016)

NON-CODING RNA

7SK dampens transcription at super-enhancers

7SK is a small nuclear RNA (snRNA) that forms ribonucleoprotein complexes (snRNPs), which are known to regulate RNA polymerase II promoter-proximal pausing; however, the genome-wide impact of 7SK snRNA and its snRNPs on transcription has remained elusive. Flynn *et al.* took advantage of state-of-the art RNA analysis techniques and found that 7SK extensively occupies transcribed genomic regions and is particularly highly enriched at super-enhancers — regulatory regions that promote high transcriptional activity. Interestingly, at super-enhancers, 7SK associated with proteins that were distinct from the ones found in the 7SK snRNP complex at promoters and specifically recruited the chromatin-remodelling BAF complex to these sites. This 7SK-mediated BAF recruitment was shown to prevent extensive transcription at super-enhancers, which often leads to convergent mRNA synthesis (occurring simultaneously at both DNA strands) and concomitant DNA damage.

ORIGINAL ARTICLE Flynn, R. A. *et al.* 7SK-BAF axis controls pervasive transcription at enhancers. *Nat. Struct. Mol. Biol.* **23**, 231–238 (2016)