IN BRIEF

DEVELOPMENT

Developmentally programmed senescence

Cellular senescence is linked to pathological contexts such as tumour suppression, as well as ageing. Now, two studies reveal that senescence contributes to mammalian embryonic development in several contexts, which suggests that it has an essential conserved role in normal physiology. Muñoz-Espín *et al.* analysed in detail the mesonephros and the endolymphatic sac of the inner ear. Senescence in both embryonic structures depended on p21 (but was independent of p53) and was regulated by the transforming growth factor-β and PI3K pathways. Furthermore, loss of cellular senescence led to developmental abnormalities (despite partial compensation by apoptosis). Storer *et al.* focused on two signalling centres in embryonic patterning — the apical ectodermal ridge (AER) and the neural roof plate. Mice deficient in p21 had defects in embryonic senescence, AER maintenance and patterning. These authors also report that senescence in the AER is instructed by the underlying mesenchyme and involves ERK signalling. Both groups find that senescent cells ultimately undergo macrophage-mediated clearance and that this is vital for development.

ORIGINAL RESEARCH PAPERS Muñoz-Espín, D. *et al.* Programmed cell senescence during mammalian embryonic development. *Cell* **155**, 1104–1118 (2013) | Storer, M. *et al.* Senescence is a developmental mechanism that contributes to embryonic growth and patterning. *Cell* **155**, 1119–1130 (2013)

TRANSLATION

When ribosomes don't stop

Typically, translation terminates when ribososmes encounter a stop codon. Stop codon readthrough results in carboxy-terminally extended nascent peptides, but its biological roles in eukaryotes have remained elusive. Here, Dunn *et al.* use a modified ribosome profiling assay to analyse genome-wide translation in *Drosophila melanogaster*. They confirm phylogenetically predicted readthroughs and identify ~300 novel C-terminal extensions, suggesting that stop codon readthrough is prevalent. The authors found that readthrough is biologically regulated during development and that C-terminal extensions produce stable protein products. In addition, they show that their peptide sequences contain subcellular localization signals, which implies that readthrough can alter protein function. Importantly, readthrough also occurred in yeast and human foreskin fibroblasts, suggesting that it has a role in several organisms.

ORIGINAL RESEARCH PAPER Dunn, J. G. *et al.* Ribosome profiling reveals pervasive and regulated stop codon readthrough in *Drosophila melanogaster*. *eLife* **2**, e01179 (2013)

CENTROSOMES

A new partner for BRCA1–BARD1

In addition to being a key factor for DNA repair, the BRCA1 (breast cancer 1)–BARD1 (BRCA1-associated RING domain 1) heterodimer affects other cellular processes, including centrosome regulation. Using mass spectrometry, Matsuzawa *et al.* identify OLA1 (Obg-like ATPase 1) as a novel binding partner of BARD1 and then show that its loss results in amplification of centrosomes. It binds directly to BARD1, BRCA1 and γ-tubulin, and resides at both centrosomes and spindle poles. Notably, the authors show that a mutation of OLA1 found in the breast cancer cell line E168Q cannot restore control of centrosome number on OLA1 knockdown. Both this mutant and a familial BRCA1 cancer mutant show loss of the OLA1 interactions with BRCA1 and BARD1, suggesting that this is physiologically important.

ORIGINAL RESEARCH PAPER Matsuzawa, A. *et al.* The BRCA1/BARD1-interacting protein OLA1 functions in centrosome regulation. *Mol. Cell* **53**, 1–14 (2013)

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